SURGERY, GYNECOLOGY AND OBSTETRICS

AN INTERNATIONAL MAGAZINE, PUBLISHED MONTHLY

VOLUME 60 APRIL, 1935 NUMBER 4

LATE COMPLICATIONS IN IRRADIATION TREATMENT OF CANCER OF CERVIX[1]

QUITMAN U. NEWELL, M.D., F.A.C.S., AND HARRY S. CROSSEN, M.D., F.A.C.S., ST. LOUIS, MISSOURI
From the Department of Obstetrics and Gynecology, Washington University School of Medicine and Barnes Hospital

THE effective treatment of cancer of the cervix uteri necessarily carries considerable risk to the patient. The risk is less with irradiation than with operation, but irradiation carries definite dangers. The serious responsibility and difficult problems in the use of radium in this situation are not generally appreciated. If the dose is too small, the distant cancer cells survive and the patient dies of recurrence. If the dosage is pushed to the point of causing devitalization of the outlying cancer cells, there is danger of irradiation injury of the rectum and bladder and the ureters.

To give the patient the best chance of cure and at the same time to hold local injury to the minimum, require expert study of the particular conditions present in the pelvis in each case, and a careful adaptation of every means to the administering of the maximum dosage permitted by those conditions. In this work, thorough knowledge of pelvic anatomy and pathology with training and experience in accurate palpation must be combined with radium knowledge and experience in order to give the patient the chance for life to which she is entitled. Hence anyone who attempts this work should have substantial training in both gynecology and radiology.

In the Washington University Medical School and Barnes Hospital, we have been using radium and X-ray therapy systematically and fairly uniformly since 1921. Our cases have been analyzed and 5 year cures and other features reported from time to time.[2] The object of this paper is to call attention to certain late complications encountered in the 371 irradiation cases treated from July 1, 1921, to January 1, 1933. The disturbances under consideration are noticed one to several years after treatment, hence the series includes only cases treated prior to 1933.

These late complications are apparently due to slow vascular changes resulting in localized tissue devitalization. Also present are slow infiltration and necrosis with resulting contraction. Considering the intensive irradiation required for this deep seated cancer, these lesions are rare in noticeable grades, as evidenced by the small number in this series. This type of lesion was found in the rectum as a stricture, in the bladder as an indolent ulcer, and in the skin as a persisting and increasing subcutaneous infiltration leading occasionally (in one case) to extensive necrosis. The amount of radium employed was approximately 100 milligrams and it was screened with ½ millimeter of silver and 1 millimeter of brass. In addition, the capsules were placed in soft rubber tubing, and distance

[2]Am. J. Obst. & Gynec., 1931, 22: 559; 1932, 24: 167; Surg., Gynec & Obst., 1934, 53: 450.

[1]Presented before the Texas Association of Obstetricians and Gynecologists, San Antonio, Texas, September 30, 1933.

screening of the bladder and rectum was secured by firm packing adapted to the particular conditions present. In most of the cases of this series, the course of treatment was started with the radium implantation. The details of the X-ray treatments are given in each case showing subcutaneous infiltration.

RECTAL STRICTURES

CASE 1. J. P., aged 44 years, in May, 1923, received 4800 milligram hours of radium for a squamous cell carcinoma of cervix of clinical stage II, League of Nations International Classification. In this case radical abdominal operation was employed, the operation being carried out a few days after the finish of the radium dosage (which was given in two treatments with an interval of 4 weeks). Following the operation the patient received deep X-ray therapy.

Two years after treatment, August, 1925, a rectal stricture was found. It was adjacent to the cervix area where the radium had been applied. It admitted the tip of the finger and was easily dilated, the walls being thin and without extensive induration. There was no indication of cancer in the rectum or elsewhere. In 1928, there was still slight narrowing but no symptoms from it. In June, 1930, there was no disturbance from the rectum or vicinity, and no evidence of cancer. In February, 1931, the patient developed symptoms of brain tumor, from which she died 4 months later. No evidence of cancer in pelvis. An autopsy was not obtained.

CASE 2. B. S., aged 46 years. In October, 1930, patient received 4500 milligram hours of radium for squamous cell carcinoma of cervix of clinical stage III, and also the usual deep X-ray therapy. In July, 1932, there was definite rectal constriction in the cervical region. This was easily kept open so that there were no troublesome symptoms, but there was still some constriction at examination in June, 1934. There was no evidence of recurrence of the carcinoma. This patient had also a second degree X-ray infiltration of the abdominal wall (see under late X-ray effects).

CASE 3. S. L., aged 43 years. In September, 1931, patient received 3000 milligram hours of radium for a squamous cell carcinoma of cervix of clinical stage III, followed by deep X-ray therapy. In a routine check-up examination in September, 1933, a rectal stricture was found opposite the cervix. It was wide and admitted the finger easily for dilatation and had given no special trouble. In April, 1934, the rectal stricture was about the same. There was no evidence of recurrence of the cancer.

CASE 4. S. G., aged 01 years. In September, 1932, patient received 4000 milligram hours of radium for squamous cell carcinoma of clinical stage III, and the usual deep X-ray therapy. Check-up examination showed no evidence of recurrence of the cancer, but in the examination in August, 1933, a rectal stricture was found. This was adjacent to the cervix, and the walls were thick and firm. The opening was small and was dilated with some difficulty. In March, 1934, the stricture was causing no trouble. It is to be noted that this stricture gave trouble about a year after treatment, while those previously mentioned were not noticed until 2 years after treatment.

CASE 5. L. F., aged 45 years. In February 1932, patient received 3000 milligram hours of radium for squamous cell carcinoma of cervix of clinical stage III, and also deep X-ray therapy. This case gives a clear indication of the etiology of these strictures. We have come to expect considerable rectal irritation in the effective irradiation treatment of carcinoma of the cervix, the rectal mucosa apparently being more susceptible to the acute effect than the bladder mucosa. Ordinarily the rectal irritation subsides after 2 or 3 weeks without further trouble. In some instances, however, the rectal irritation progresses to ulceration and infiltration with subsequent stricture formation.

In this case the rectal irritation persisted and after 3 months (in May, 1932) there was definite ulceration. This gradually healed, but with evident beginning stricture from the scar tissue. This emphasizes the importance of investigation and treatment of any persisting rectal symptoms, and also of rectal palpation in the check-up examinations of these cancer patients. This patient had also a first degree X-ray infiltration of the skin of the abdominal wall (see under late X-ray effects).

BLADDER ULCERATION

CASE 1. B. H., aged 45 years, in June, 1923, received 3800 milligram hours of radium (in two treatments close together) for squamous cell carcinoma of cervix of clinical stage late II, and also the regular deep X-ray therapy. Patient remained in good health for 3 years (until August, 1926), when she complained of bladder symptoms and the passing of bloody urine. Cystoscopic examination revealed a small ulcer in the bladder opposite the cervical region. This healed promptly under silver nitrate irrigations, and the bladder symptoms disappeared. There was no evidence of recurrence of the cancer. Subsequent check-up examinations showed no evidence of recurrence of the cancer and no bladder disturbance. In an examination in June, 1933, the patient showed a marked hypertension, and in December she died of apoplexy. No autopsy was obtained.

CASE 2. E. P., aged 33, in September, 1933, received 3000 milligram hours of radium for squamous cell carcinoma of cervix of clinical stage II. The patient received also deep X-ray therapy, of which details will be given later as this is the patie with

sloughing of the skin mentioned under late X-ray complications. In March, 1928, this patient complained of bladder symptoms, and on vaginal palpation there was a firm infiltration involving the bladder wall near the cervix. Cystoscopic examination showed just beyond the trigone an area of elevation, varicosities, submucous hæmorrhage, and a small ulcer. It gave the impression of chronic inflammation rather than malignancy, and the subsequent course confirmed this diagnosis. This bladder disturbance cleared up under irrigations and instillations. The patient has been kept under observation, the last examination being in June, 1934, and at no time has there been any evidence of the malignancy for which she received treatment in 1925.

Case 3. R. H., aged 64, in June, 1926, received 4000 milligram hours of radium for squamous cell carcinoma of cervix of clinical stage III, and the usual deep X-ray therapy. Patient remained well until January, 1933, when she developed bladder distress and bloody urine. Cystoscopic examination showed an area of partial devitalization about 2 centimeters in diameter just above the level of the ureteral orifices. Surrounding the area were numerous dilated blood vessels radiating somewhat like the spokes of a wheel. Figure 1 (frontispiece) gives a very good representation of the cystoscopic appearance. The vessels bled easily when traumatized.

A small piece of tissue was removed from the area by Dr. J. R. Caulk, who had charge of the cystoscopic work in these cases. Microscopic investigation of the removed tissue showed chronic inflammation, but no malignancy. Two fulguration treatments stopped the bleeding, and the bladder irritability gradually lessened. In June, 1934, there had been no more bloody urine and the bladder symptoms were greatly improved.

Case 4. A. S., aged 46 years, in August, 1929, received 3600 milligram hours of radium for adenocarcinoma of cervix of clinical stage III, and the regular deep X-ray therapy. The patient had also myoma of uterus and exophthalmic goiter, and received medical treatment for the latter. The patient was in good health until July, 1932, when she began to have bladder distress and passed bloody urine.

Cystoscopic examination revealed numerous dilated blood vessels in the portion of bladder opposite the cervix. Between the vessels, the bladder wall was pale and, in the center, was a grayish area. The entire field involved was about 3 by 4 centimeters, and it bled easily when touched. Scrapings removed through the cystoscope showed chronic inflammation but no malignancy. The bleeding was easily controlled by irrigations and instillations. Check-up examinations in July and September, 1933, showed absence of any further bleeding and not much bladder disturbance. At no time was there evidence of recurrence of the cancer for which treatment was given in 1929.

Of these 4 cases of late bladder complications, the interval between irradiation treatment and appearance of bladder trouble was 3 years, 2½ years, 6 years, and 3 years, respectively. The bladder symptoms developed rather suddenly and were soon followed by bloody urine. Cystoscopic examination showed a fairly uniform picture consisting of a devitalized area or ulcer with radiating dilated blood vessels (Fig. 1, frontispiece). The area bled easily when traumatized. Specimens removed through the cystoscope showed chronic inflammation without malignancy. In these particular cases the subsequent history showed the absence of accompanying malignancy, though such a non-malignant lesion might occur in a case showing recurrent cancer activity either at that time or later. The lesion and resulting bladder symptoms yielded fairly well to irrigations and instillations, fulguration to stop bleeding being required in only one case.

SLOUGHING OF SKIN

Pigmentation of the skin is, of course, to be expected over portals of entry for therapeutic roentgenization and is not counted of pathological significance. The late complication here referred to is a marked subcutaneous infiltration, which caused thickening and irregularity of the skin and was later followed by sloughing.

Case E. P., aged 33 years, in September, 1925, received 3000 milligram hours of radium for squamous cell carcinoma of cervix of clinical stage III, and also deep X-ray therapy. The X-ray therapy was given in treatments on September 9, 1925; November 4, 1925; January 18, 1926; March 23, 1926; May 7, 1926; July 25, 1926; and November 26, 1926. Each dose (given in two areas—one front and one back), milliamperes 30; time, 13 minutes; kilovolts, 200; filter, 1 millimeter copper, 1 millimeter aluminum; portal 20 centimeters; distance 50 centimeters.

Check-up examinations from time to time showed the pelvis clear of evidence of recurrence of the cancer. As early as the latter part of 1926 it was noticed that there was an infiltration of the skin over an area of about 6 by 7 centimeters on the lower abdomen just above the symphysis pubis. Besides being highly pigmented, the skin was somewhat œdematous, with a deep, firm infiltration. There was a smaller area presenting similar characteristics posteriorly over the sacrum. In October, 1927, the subcutaneous infiltration had increased and definite telangiectasis was appearing in the areas. Figure 2 (frontispiece) gives a very good idea of the condition of the abdominal wall at this time. In March, 1928,

the patient developed an ulcer of the bladder, which is reported as Case 2 under bladder ulceration. This yielded to treatment as previously mentioned. The abdominal wall infiltration showed increasing telangiectasis and serious local irritation. Figure 3 (frontispiece) shows the condition of the abdominal wall at this time.

The patient disappeared and did not return until October, 1932. As the reason for not returning, she stated the skin of the lower abdomen sloughed away, keeping her in bed for a period of 18 months. Apparently an area about the size of the hand in the involved region of the lower abdomen had necrosed. The necrosed skin had sloughed away to the subcutaneous tissue, leaving a large granulating surface. The patient was very stout with overhanging abdominal wall, and the granulating surfaces, being approximated, had healed together. At the visit in October, 1932, the process of healing was only partly completed. There was still a small granulating area in the right side, and the healed area was tender. On her return, September 11, 1933, the abdomen was entirely healed. The line of scar tissue is shown in the photograph in Figure 4. In the right side of the scar, there was an infiltrated area forming a small nodule. This was excised for microscopic investigation, and showed chronic inflammation without malignancy. Over the sacral area there was much infiltration and vascularization, but no sloughing. The photograph in Figure 5 is to show this area of discoloration. Naturally we have been watching the abdominal surface for malignant change, but so far none has appeared. At the last examination in June, 1934, the patient was in good general health, and up and about her work without serious discomfort. At no time has there been any evidence of recurrence of the cancer for which she received treatment in 1925.

As to the etiology of this complication—why it appears in an occasional individual and not in others—the idiosyncrasies of the patient with the special reactions of her skin and subcutaneous tissues and blood and lymph must be taken into consideration. It is to be hoped that the study of this and allied conditions will develop some method of spotting those individuals with handicaps which limit their capacity to stand deep roentgenization.

In the course of check-up examinations we have noted two lesser grades of what appears to be this same process. One case showed a very mild change in the skin and subcutaneous tissue.

This patient, aged 45, was given radium and deep X-ray therapy in February, 1932. She developed a marked proctitis with some ulceration and later a rectal stricture, which is reported as Case 5 under rectal strictures. The X-ray therapy consisted of 1030 r to lower abdomen on February 16, 1932, and 850 r to lower back on February 17, 1932. May 19, 1932, the patient complained of pain in the lower abdomen and vulva and thighs. Examination showed the abdomen markedly pigmented below the umbilicus with absence of pubic hair. No special vascularity was seen. There was some deep infiltration, causing thickening and irregularity of the skin. In June, 1934, the condition remained about the same.

Another case showed what may be designated as the second stage of the process in which there was obvious vascular change in the form of telangiectasis.

This patient, aged 46 years, received radium and X-ray therapy in October, 1930. She developed a mild rectal stricture, and is reported as Case 2 under rectal strictures. The X-ray therapy consisted of 900 r to lower abdomen on September 30, 1930, and 1000 r to lower back on October 1, 1930.

In the examination in June, 1932, it was noted that there was chronic infiltration of the abdominal wall with some superficial vascularity. By December, 1932, the condition of the wall was definitely worse, the skin being raised in parallel ridges. In September, 1933, the process showed no further progress, giving the impression that it had reached its maximum. June, 1934, the telangiectasis was more marked, pain in the abdominal wall was increasing, and the condition was becoming definitely worse.

GENERAL PLAN OF IRRADIATION TREATMENT

In order to improve the follow-up in our cancer cases and better co-ordinate the work of the various services required in their treatment, we established some years ago a gynecological cancer clinic. This clinic is held every Tuesday afternoon and all cancer cases of the general gynecological clinic are referred to it. A special history is made in each case, and a careful description of the growth recorded. The clinical classification of the cancer is made as far as conditions will permit, placing it in stage I, II, III, or IV of the League of Nations International Classification. A specimen is taken and sent to the laboratory for microscopic investigation and a bacterial culture also is taken from the growth, as a control in clearing up the pyogenic infection which is usually present. Antiseptic douches are begun, to be continued while the special treatment for the cancer is being carried out. The patient is immediately turned over to the

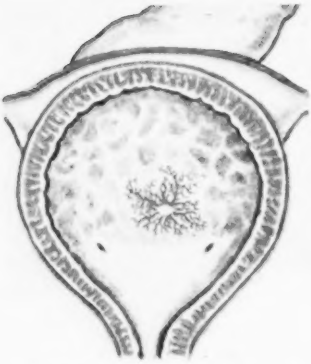

Fig. 1. Late devitalization ulcer of bladder, appearing 6 years after irradiation treatment of carcinoma of cervix uteri. The grayish appearance with numerous dilated blood vessels radiating toward the center is typical of this late devitalization lesion. It is located opposite the cervix region, may appear from 2 to several years after the irradiation, and yields readily to treatment.

Fig. 2. Late subcutaneous infiltration of abdominal wall, appearing 2 years after irradiation treatment of carcinoma of cervix. Third degree, with deep extensive infiltration and marked widespread vascularization of the surface.

Fig. 3. The same abdomen as shown in Figure 2, but 5 months later. The devitalization had definitely progressed, and there was a small bluish area which appeared to be beginning necrosis.

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Fig. 4. Photograph of the same abdomen shown in Figure 3 (frontispiece), but 5 years later. An area the size of a hand had sloughed to the subcutaneous tissue. The granulating surfaces of the lax wall had fallen together and united. Microscopic examination of a section removed from the suspicious area on the right side showed no malignancy.

Fig. 5. Same patient as in Figure 4. Back view, to show the discoloration of the skin over the sacral area, where there was much infiltration and vascularization, but no sloughing.

social worker, and it should be emphasized that a good social worker is very important. She sees these ward patients in the clinic and hospital and, after dismissal from the hospital, sees that they return to the cancer clinic at the appointed time. She makes all appointments for X-ray treatments with the Department of Radiology. The Department of Radiology has full charge of the deep X-ray therapy, and works in close co-operation with the gynecologist. The gynecologist has full charge of the radium treatment, and makes all radium implantations.

The systematic course of irradiation extends over a period of about 3 months. It is usually started with deep X-ray therapy, in order to secure some devitalizing effect on the outlying cancer cells before the stir-up of the radium implantation in the center of the mass. This preliminary X-ray therapy and course of local antiseptic treatment before the radium implantation, is especially important where there is associated infection, as there usually is with ulceration or papillary formation. Two or 3 weeks after the preliminary roentgenization, the patient is hospitalized for the radium treatment.

After accurate implantation of the radium, the bladder and rectum are pushed away by a very firm packing, in order to permit the maximum radium dosage which can be safely given with the conditions present in that pelvis. The packing is so firm that the patient can not urinate and, on this account and also to keep the bladder empty and away from the radium, a retained catheter is used. The maximum dosage which can be given safely depends largely on how far away the bladder and rectum can be pushed by the packing. This depends on the amount of fixation of these structures by the carcinomatous infiltration, and of course varies in each case. As a rule the infiltration is mainly into the lateral parametrial tissues, with only late involvement of the bladder and rectal walls. At the time of the radium implantation, a careful examination under analgesia is made and a definite decision reached and recorded as to the clinical classification of the case. It is well also to make another bacterial culture at this time. For the radium treatment, the patient ordinarily remains in the hospital about 5 days, and after discharge returns to the clinic for check-up examinations. About 8 weeks after the radium treatment the patient is given the second series of deep X-ray.

This 3 months course of treatment comprises the regular course. Additional X-ray or radium treatment is given only on special indication. After the 3 months' course the patient returns for check-up examination

about every 2 months the first and second years, every 3 months the third year, every 4 months the fourth year, every 6 months the fifth year, and once a year after that. Of course, if there is any local disturbance between times, the patient is to come at once.

CONCLUSIONS

In the intensive irradiation treatment of 371 patients with carcinoma of the cervix, most of them advanced, we have encountered the following late complications:

Rectal stricture: 5 cases, mostly mild and all yielding satisfactorily to dilatation treatment.

Bladder ulceration: 4 cases, all responding to irrigations and instillations, and only 1 requiring fulguration to stop bleeding.

Sloughing of the skin: 1 case of extensive sloughing with subsequent healing. There was also 1 case of subcutaneous infiltration (first degree) which seemed stationary, and 1 case of subcutaneous infiltration with superficial vascular change (second degree), which was definitely progressive.

Of course, some mild rectal strictures and lesser grades of subcutaneous infiltration may have escaped observation, but the more serious lesions are not likely to be missed. We feel that we have been very fortunate to have accomplished so much in this disease, with so few serious complications. This applies both to the radium and the X-ray therapy. The radiologist, Dr. Sherwood Moore, has succeeded in giving massive doses of X ray, which aided materially in curing patients and in giving temporary relief to others, with very few of the serious late effects. However we are always seeking improvements, and one of the problems of the day is the difficult task of diminishing still further the incidence of such complications.

THE CAUSE OF DEATH DUE TO LIVER AUTOLYSIS[1]

EDWARD C. MASON, M.D., PH.D., AND CARL A. NAU, M.A., M.D., OKLAHOMA CITY, OKLAHOMA
From the Department of Physiology, University of Oklahoma, School of Medicine

CONSIDERABLE confusion has developed in an attempt to explain the cause of death when liver tissue is found free within the abdomen. The observations presented by the various studies on this subject are often quite contradictory; however the several authors state that one of the three following factors is the cause of death: (1) a toxic substance generated in the autolyzing liver (4, 5, 6, 1); (2) the presence of bacilli resembling the Bacillus welchii (3); (3) anhydræmia due to the loss of fluid into the abdominal cavity (7).

We have continued our studies in order to determine if the type of diet could in any way modify our previous results. While these studies were being conducted we have been making additional observations which we believe furnish a satisfactory explanation for the cause of death.

THE EFFECT OF HERBIVOROUS DIET

Rabbit 1. Female, weight 8.25 pounds. Nembutal anæsthesia 1.6 cubic centimeters (1 grain per cubic centimeter) intraperitoneally, supplemented with ether. October 23, 8:40 p.m. under aseptic precautions 16.5 grams of liver was removed from a second rabbit and placed high in the abdominal cavity. October 24, animal had quiet day. Water consumption in 24 hours was 500 cubic centimeters. October 25, 8:30 a.m., animal was very active and alert. November 6 (14 days after operation), weight 8.25 pounds. The rabbit was killed and postmortem examination was made.

Rabbit 2. Female, weight 7.25 pounds. The procedure the same as for rabbit 1 was used. October 27, 30 grams of liver including the gall bladder placed in the abdominal cavity. October 28, animal quiet but alert. October 29, animal was alert and active. November 6, 10 days after operation, animal weighed 6.75 pounds. It was killed and a postmortem examination was made.

Rabbit 3. Male, weight 7.75 pounds. Same procedure as in previous experiments was used. November 4, 42 grams of liver was placed in the abdominal cavity. November 6, animal was alert, active, and taking food and water. November 6, 56 hours after operation, animal weighed 7.5 pounds. It was killed and postmortem examination was made.

[1] Received for publication July 31, 1934.

The results of these experiments do not agree with the findings of Andrews and Hdrina (1) who state: "The influence of diet was then studied by using herbivorous animals and the liver of 1 rabbit was planted into 5 other rabbits and exactly the same clinical picture of autolytic peritonitis was produced."

Dog 1. Female, weight 24 pounds. Preliminary preparation consisted in a high carbohydrate, low protein diet for 3 weeks. At 8:00 p.m., nembutal 3.4 cubic centimeters (1 grain per cubic centimeter) was given. At 8:45 p.m., this was supplemented with ether, and 30.5 grams of liver was removed and placed high in the abdominal cavity. At 10:00 p.m., the condition was good. At 7:45 a.m., the animal was dead, 10 hours after operation.

Dog 2. Female. High carbohydrate, low protein diet was given for 5 weeks and 3 days. At 9:30 p.m., ether anæsthesia was administered, and 46.75 grams of liver was sectioned and placed in the abdomen. At 10:00 p.m., the dog was in good condition. At 11:10 a.m., the animal was dead, 13 hours, 40 minutes after operation.

These two dogs, fed on a high carbohydrate diet, showed no additional resistance; they lived only 10 hours and 13 hours 40 minutes, respectively, following the operation. These are probably the most sudden deaths we have recorded.

THE EFFECT OF DOG LIVER IN RABBITS AND RABBIT LIVER IN DOGS

Confronted with the fact that rabbits survived the operation while dogs on a similar diet did not, we next attempted to learn if the rabbits' resistance was due to the increased resistance of their intact liver or due to the fact that the sectioned liver did not yield such toxic substances.

Rabbit 4. Weight 6.25 pounds. Nembutal and ether anæsthesia was used. At 10:40 a.m., 23 grams of dog liver was removed while the animal was alive, and placed in the abdomen of the rabbit. At 11:00 p.m., animal was alive but not in good condition. It was found dead the next morning.

Rabbit 5. Weight 7.75 pounds. Same anæsthesia as for rabbit 4 was used. At 10:00 a.m., 21 grams of fresh dog liver was placed into the abdomen. At 5:15 p.m., breathing was labored and rabbit was

Fig. 1. Rabbit liver tissue undergoing aseptic autolysis.

very listless. It was found dead the next morning.

Rabbit 6. Weight 8.25 pounds. The same anæsthesia was used as in previous experiments. At 10:25 a.m., 29 grams of fresh dog liver was placed in the abdomen. At 5:15 p.m., the rabbit was alert and appeared well. It was found dead the next morning.

Rabbit 7. Weight 8.25 pounds. The same anæsthesia as formerly was used. At 10:15 a.m., 18.25 grams of fresh dog liver was placed in the abdomen. At 1:15, the animal was sitting up and was alert. At 5:15 p.m. animal was apparently well. It was found dead the next morning.

Dog 3. Male. Weight 33 pounds. Nembutal and ether anæsthesia was used. At 7:30 p.m., 73 grams of liver was removed from a rabbit while it was alive and placed into the abdomen of the dog. At 8:45 a.m., the dog was standing. It drank much fluid during the day, the intake being approximately 1210 cubic centimeters above the output. At 7:00 a.m. (second day), the dog was found dead. Death occurred about 30 hours after operation. At 9:30 a.m., postmortem examination revealed the usual findings with 300 cubic centimeters of fluid in the abdominal cavity.

THE RELATIVE TOXICITY OF IMPLANTED CENTRAL AND PERIPHERAL LIVER TISSUE

Desiring to obtain additional support for our original observation, in which we found that rabbits survived the operation, we extended our series to include 3 more rabbits. However, in order to use fewer animals we removed the entire liver of 1 rabbit and divided it into three approximately equal portions. One piece was then placed into the abdomen of each of 3 animals with the following results.

Rabbit 8. Weight 8.25 pounds. At 10:30 a.m., 37.5 grams of liver was inserted. At 7:50 a.m., the next day, the animal died.

Rabbit 9. Weight 6 pounds. The animal died the morning after 38 grams of liver had been inserted.

Rabbit 10. Weight 7.25 pounds. The animal died the morning after 41.5 grams of liver had been inserted.

Fig. 2. Rabbit liver tissue undergoing aseptic autolysis. Greater magnification than used in Figure 1.

These findings are contrary to our original observation and therefore agree with those reported by Andrews and Hrdina (1). It will also be noted that our technique in this series of rabbits was the same as that employed by Andrews and Hrdina; that is, introducing the entire liver of one animal into the abdomen of several other animals. Analysis of these results suggested to us that the only difference in the procedure used in the surviving animals and those that died, was the difference in the location from which the implanted liver was selected. In the former animals we introduced liver sectioned from the periphery while in the latter the sectioned liver included the central portion.

The following experiment illustrates the fact that implanted liver taken from the peripheral portion is less toxic than that taken from the central portion.

Rabbit 11. Weight 8.25 pounds. At 8:15 p.m., 15.9 grams of liver (peripheral portion) removed from a second rabbit and implanted. At 12:00, noon, the following day, rabbit was quite active and remained in good condition. It was killed 67 hours following operation and postmortem examination was made.

Rabbit 12. Weight 7.25 pounds. At 8:30 p.m., 15.9 grams of liver (central portion) was removed

Fig. 3a. The host's liver (rabbit) accompanying the presence of aseptic autolysis.

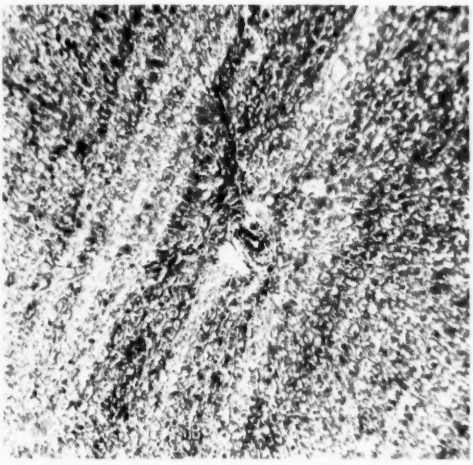

Fig. 3b. The host's liver (rabbit) accompanying aseptic autolysis.

from a second rabbit and implanted. At 8:15 a.m., the following day, condition was good. At 10:30 a.m., animal died suddenly. Condition appeared good 10 minutes before death. Postmortem examination was made.

A STUDY OF THE HISTOLOGICAL AND BACTERIOLOGICAL CHANGES

The animals which survived the operation, that is, those which received peripheral liver tissue, were subsequently killed and an autopsy performed. The examination revealed a postmortem condition quite different from that which we have previously found (4) in the non-surviving animals. The implanted liver is firmly adherent to a loop of bowel, it is creamy-green in color, contains no gas and no odor. Occasionally, there is present only a small amount of free fluid in the peritoneal cavity. The sectioned liver shows a marked loss of weight ranging from 24 to 54 per cent in 10 days and 14 days, respectively. This loss of weight is the result of phagocytic action and is well illustrated in the microscopic sections.

Figure 1 is an enlarged photograph of a paraffin section of liver tissue undergoing aseptic autolysis. It will be noted that the invading phagocytic cells are arranged in a dense formation producing a definite line below the liver capsule.

Figure 2 is a photomicrograph of the section in Figure 1 and shows the process more in detail.

While the autolysis illustrated is taking place in the implanted liver, the hosts' organs are also definitely damaged. Accompanying such damage the animals showed definite toxic symptoms.

Sections of the host's liver and kidney are presented in Figures 3a and b and Figure 4. Dr. L. A. Turley of the Department of Pathology gives the following description of the liver and kidney. This description is confined to tissue obtained by killing the rabbits ten days postoperative.

Liver. "Cords of central and middle zones of the lobules show rarefaction so marked that the nuclei of the cells appear to be held in place by a lace-like web. The cells of the peripheral zones have a fine, foamy appearance. The tissues of the portal canals are infiltrated with lymphocytes, endothelial cells, and an occasional polymorphonuclear leucocyte. This infiltration is most marked around the bile ducts. This liver is the victim of what appears to be a rather acute and marked fatty degeneration plus a septic cholangeitis."

Kidney. "The lesions in the kidney vary with the different fields. In some fields the capillaries of the tufts and tubules are en-

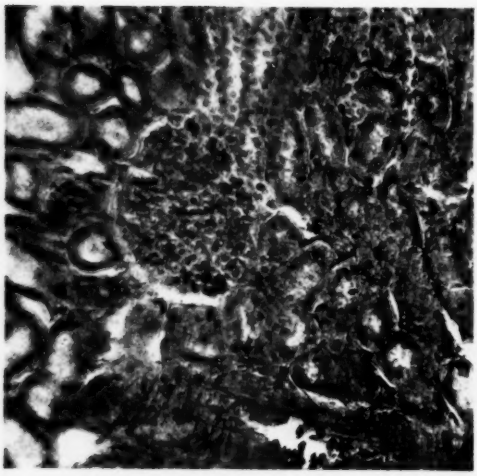

Fig. 4. The host's kidney (rabbit) accompanying aseptic autolysis.

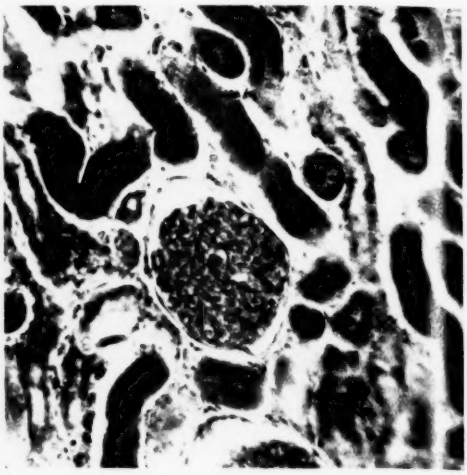

Fig. 5. Photomicrograph of frozen section of the kidney of a non-surviving dog.

gorged with red blood cells, the tufts are swollen until they fill the subcapsular spaces, the tubule epithelium is swollen, and the inner margins are frayed. The lumen contains granular débris. In other fields the glomerular tufts are approximately normal in size, leaving a fair subcapsular space. The tubular epithelium shows some erosion, but also many of the cells show the brush-like inner border. This kidney appears to be recovering from a rather intense, toxic parenchymatous nephritis. The cause of the condition is still operating though in lessened intensity as is shown by the areas of present, acute nephritis."

From the changes described it is evident that aseptic autolysis is associated with a definite tissue reaction on the part of the host. Although the animals showed marked toxic symptoms during such changes, there was not sufficient destruction to cause death.

Microscopic frozen sections of liver of a non-surviving dog reveals the following.

Liver. "The portal veins are engorged with red blood cells. The sinusoids are full of red blood cells, especially in the central zones. An occasional polymorphonuclear leucocyte is seen in the sinusoids. There is some hypertrophy of the Kupfer cells. The portal canal tissues are infiltrated with lymphocytes and a few polymorphonuclears. The liver cords are somewhat swollen. There is a fibrinocellular exudate on the surface of the liver capsule, with beginning invasion of the underlying tissues. The cells in the exudate are 'polys,' endothelial cells and some lymphocytes. The liver is suffering from a septic perihepatitis, and is receiving excess blood from a septic area via the portal circulation. As yet, no severe parenchymatous change has taken place."

Figure 5 is a photomicrograph of a frozen section of the kidney of a non-surviving dog.

Kidney. "The glomerular tufts are swollen until they fill the subcapsular space. All capillaries of the tufts and tubules are engorged with red blood cells. The tubular epithelium is swollen until the lumina are reduced to mere slits or are not discernible. There is some lytic degeneration of the papillary duct epithelium. This is a case of severe, acute, toxic nephritis in the congestive stage."

In all animals which died, both rabbits and dogs, an organism was constantly found. The rôle of the organism in the chain of events which leads to death is illustrated by the following experiment.

Rabbits 13 and 14. A small portion of the autolyzed liver was removed from rabbit 12 (at the time of death) and ground with salt solution. Two cubic centimeters of solution was injected intravenously

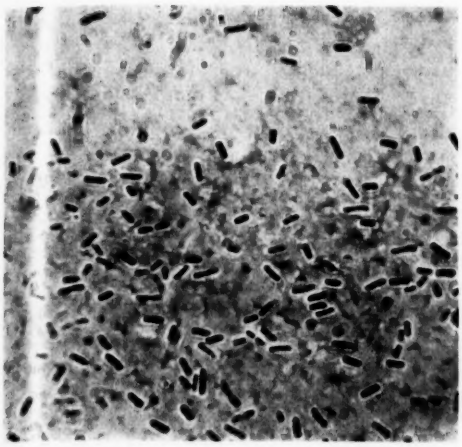

Fig. 6. Organism obtained from central portion of rabbit liver.

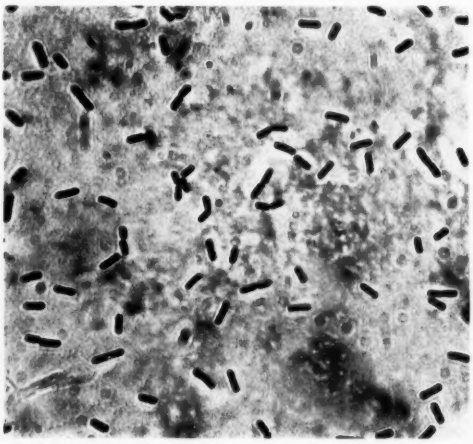

Fig. 7. Impression smear of the liver from incubated rabbit 13.

into each of rabbits 13 and 14. After 5 minutes rabbit 13 was killed and placed in an incubator for 22 hours. Autopsy at this time showed the following: The liver was soft and friable with a consistency that of soft cottage cheese. All the tissues were crepitant. The striated muscle was riddled and the odor was very foul. Impression smears were made and stained by the Gram's method which showed enormous numbers of positive rods identical with those found in the autolyzed liver of rabbit 12.

Rabbit 14. Was permitted to live and showed no ill effects from the injection.

Figure 6 is a photomicrograph of the organism which was obtained from the culture of the central portion of liver, part of which was implanted in rabbit 12. The peripheral portion of the same liver and that which was introduced into rabbit 11 on culture showed no anaerobic organisms.

Figure 7 was obtained as an impression smear from rabbit 13. The source of the organism was the same as that reported in Figure 6.

DEDUCTIONS

Sectioned liver tissue undergoing aseptic autolysis within the abdomen is accompanied by definite damage to the host. This is evidenced by changes in liver and kidney of host and also by toxic symptoms manifested.

In all of our previous reports on this subject, the experimental data were obtained from dogs. Therefore, when the first 3 rabbits of this series survived the operation, we were of the opinion that the survival was due to the type of diet selected by the rabbit. This conclusion also agreed with the observation of Salzmann who found that rats either survived the operation or died depending entirely upon the type of diet fed the animals.

Continuing our experiments we have been forced to conclude that all the surviving animals received implanted liver tissue sectioned from the periphery of the liver. Such tissue proved to be free of the anaerobic bacillus. We further observed that the implanted liver tissue sectioned from the central portion caused death and also gave a positive culture for the organism. It has been observed by Andrews et al (1) and also by Dvorak that this organism may be injected in large amounts into the peritoneal cavity without causing the death of the animal. We have injected the organism intravenously and found it affected the animals but little, if at all. However, if shortly after such an injection we killed and incubated the animal, we subsequently found the tissues completely riddled by the organism, the liver being destroyed.

The sequence of events which leads to the death of the animals appears to be as follows: The bacteria remain in a latent state until stimulated to activity by the asphyxiation of the liver tissue. The activity of the bacteria then accelerates the production of toxic prod-

ucts and these, in turn, cause death by producing a chemical reaction. The picture is not that which we usually consider as a generalized peritonitis but the bacteria confine their activity locally to the selected substrate. Also, the rapidity with which death follows the operation, after 10 to 15 hours, is not in keeping with the usual picture of peritonitis. The damage to the tissues of the host shown in the microscopic sections, with and without the presence of bacteria, rather suggests that the process which produces death is not necessarily a difference in kind but rather a difference in degree; the action of the bacteria only accelerating the liberation of toxins from the liver tissue. Trusler and Reeves have demonstrated the fact that the organism does not produce exotoxins and it has also been shown that the organism may be cultured within the abdomen, substrates other than adult liver tissue being used without causing death (3).

CONCLUSIONS

1. Additional evidence is submitted to support the contention that the absorption of dead and autolyzed tissue is associated with toxic symptoms on the part of the host.

2. Death is not due to a generalized peritonitis but rather to the absorption of toxic products generated from the liver tissue deprived of its circulation.

3. The anaerobic bacillus found at the time of death may be injected intraperitoneally and intravenously without causing any harmful results.

We wish to express our appreciation for the assistance given us by Miss Ida Lucille Brown of the Department of Bacteriology.

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THE HISTOLOGY OF THE BILIARY DUCTS AND ITS CORRELATION WITH THE SYMPTOMATOLOGY OF COMMON DUCT STONE[1]

IAN G. MACDONALD, M.D., C.M., CORNWALL, NEW YORK

STANDARD works of reference in medicine and surgery enumerate a remarkably uniform list of symptoms said to be caused by the presence of calculi in the extrahepatic biliary passages. In the past several years, the "asymptomatic common duct stone" has been the theme of several writers (3, 6), but nevertheless the gathered opinions of members from several surgical staffs gave the impression that biliary colic is the chief signpost to a diagnosis of calculus in the common duct. In direct variance with this is a statement by Professor D. P. D. Wilkie, of Edinburgh, that common duct stone never gives rise to colic, since that section of the biliary tract exhibits in its wall no muscular tissue, and is therefore non-contractile, non-peristaltic. Attacks of biliary colic, Wilkie stated, are produced only by the passage of a stone along the cystic duct with its regular layers of musculature. This assertion seemed sufficiently novel to merit investigation, and if proved would obviously be of great diagnostic importance. The subject has been pursued along three lines:

1. Review of recent literature.

2. Study of the detailed histology of the bile ducts.

3. Analysis of case histories of patients proved to have had choledocholithiasis over a 5 year period at the Los Angeles County General Hospital and the Toronto General Hospital.

It will be shown that the correlation of anatomical findings with symptoms in these cases, with due reference to the duct histology outlined below, disprove practically all the commonly accepted explanations of pain production in biliary colic.

1. Much of the recent literature on the subject has been concerned with the absence of the classical signs and symptoms in many cases, and the wide variance possible in the clinical picture. In 1877, Charcot first described the intermittent fever in common duct obstruction which has since been associated with his name. Kehr, in 1900, remarked that jaundice may be absent in common duct stone, and in Jordan's series of 106 operations for this condition 10.6 per cent of the patients were without jaundice. Oertel described a case in which complete common duct obstruction by stone had been compensated for by saccular dilatations of the ducts to such an extent that the patient died without jaundice. Mann's experiments verified this possibility in animals, where dilatation of the ducts delayed the onset of jaundice. Clute reported that 31 per cent of his operative cases with common duct stone showed no jaundice. He recommended more frequent choledochotomy when operating for calculous cholecystic disease, having found stones in the common duct in 18 per cent of his cases in a 3 year period due to this procedure. He remarked that a slight elevation of blood bilirubin often means common duct stone. A further series of cases reported by Klingenstein showed 6 per cent without jaundice, and a slightly smaller percentage without colic.

The theories that have been advanced to account for causation of biliary colic are many and varied. Such are concerned with impaction of a calculus in the ampulla of Vater, distention of the ducts, and the size and shape of the obstructing stone. The cases to be quoted below will suggest the futility of such explanations. Rolleston and McNee are authorities for the statement that severe biliary colic may be present in the absence of any calculi, being due to cholecystitis with inflammatory reaction in the sensitive subperitoneal tissues. Most commonly accepted is the view that colic is due to spasm of the ducts by their constituent muscular layers. With this in mind the investigation of the histology of the bile ducts was undertaken.

2. An amazing variance of ideas as to the normal histology of the bile ducts was encountered in the various standard works, old

[1]From Department of Surgery, University of Southern California; and from work begun in Department of Surgery, University of Toronto.

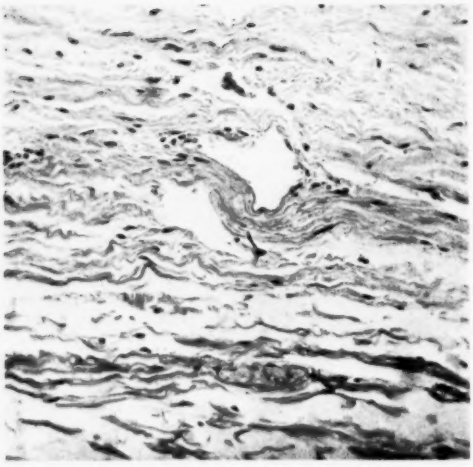

Fig. 1. Low power field from an area in a longitudinal section of the common bile duct, 2 millimeters distal to the sphincter of Oddi. Shows scattered muscle fibers, usually lying individually, among fibrous and elastic tissue. At the lower edge are seen a few organized muscular strands tailing off from the sphincteric muscle. Phosphotungstic acid stain.

and recent. For example, a large volume of *Special Cytology*, published in 1928 (10), states that in the cystic duct exist three-directional smooth muscle fibers, while in common and hepatic ducts only very small amounts are present, and without definite layers. Maximow emphasizes the fact that only the common duct shows bundles of smooth muscle running in oblique and longitudinal directions, forming an incomplete layer around the duct. Burden observed that the hepatic, cystic, and common bile ducts are identical in structure, there being bundles of unstriped muscle lying in the outer layer of areolar tissue together with blood vessels and lymphatics. He claimed that these muscle bundles were well developed and formed isolated longitudinal and circular layers, thus producing a loose network rather than a compact layer. Rolleston and McNee make the bald statement that the bile ducts are without muscular tissue at any point, but quote no proof of such statement. In spite of the fact that nearly all possible combinations seemed to have been exhausted by these various authorities, it was proposed to prove, if possible, which of the statements here quoted was correct.

Microscopic sections were first made from the various parts of the bile duct—hepatic, cystic, and common. Three stains were employed: hæmatoxylin and eosin, phosphotungstic acid stain for muscle, and Weigart's for elastic tissue. As no muscular coats could be found in the bile ducts in the material examined from the first few autopsies, a more systematic method was followed.

The gall bladder and the cystic and common ducts down to the entrance of the latter into the duodenum and including a section of the duodenal wall, were removed from cadavers showing no gross evidence of biliary tract infection. After being washed in water the preparation was injected through the ampulla of Vater with Zenker's solution until the ducts were moderately distended. When sufficiently fixed, sections were taken at regular levels, as follows:

A. Through sphincter of Oddi and along the terminal portion of the common duct.
B. From proximal portion of common duct.
C. From distal end of cystic duct.
D. From proximal portion of cystic duct.
E. Through the junction of cystic duct with the neck of the gall bladder.

These sections were then stained with phosphotungstic acid, which, after study of numerous sections, was determined to be the best stain for the detection of muscle tissue. In all, 84 sections, some transverse, but most of them longitudinal, were prepared from the material obtained from 14 autopsies.

A study of these sections shows that the muscular coats, as such, cease very abruptly at the neck of the gall bladder, sometimes with a sphincter-like structure at the origin of the cystic duct. The remainder of the biliary ducts, common, cystic, and hepatic, are merely fibro-elastic tubes, lined with a high columnar mucosa, covered by a serosal layer with the usual subserosal areolar tissue. Only small isolated fibers of unstriped muscle are present, sometimes indistinguishable from connective tissue cells. In many sections, a search of several high power fields is necessary to demonstrate even isolated muscle cells. A striking point in the study of many of these ducts is the presence of comparatively large glands in the duct walls, as described by Burden. It is a

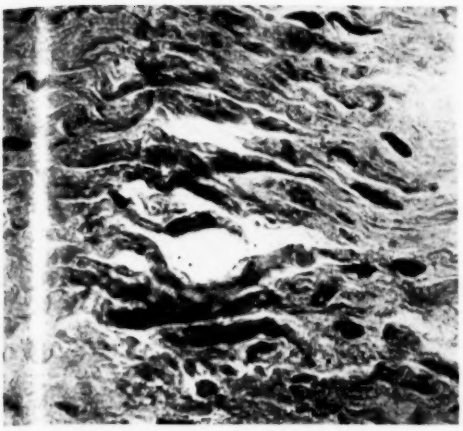

Fig. 2. High power view of a field from Figure 1. Individual muscle fibers stand out in contrast to connective tissue.

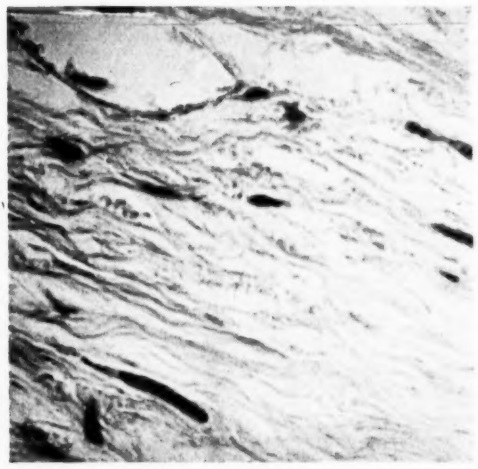

Fig. 3. High power view of an area several low power fields distal to that in Figure 1. Note marked decrease in number of muscle fibers. Phosphotungstic acid stain.

real possibility that these glands offer lodging to organisms metastatic from other foci of infection, and subsequently become responsible for low grade cholangitis. Nerve fibers are present in great abundance, usually distributed in the outer part of the wall.

3. The clinical review was confined to those cases definitely proved to have had calculi in the common duct. This reduced the number of cases in the 5 year period to 16 at the Toronto General Hospital (1926–31), and 8 at the Los Angeles County General Hospital (1927–32), in which stone had actually been removed from the common duct at operation or autopsy. When contrasted with the figures from other surgical clinics (Mayo Clinic 106 in 10 years; Mayo-Robson 380 in 10 years) this seems to indicate an extraordinarily low incidence of calculous cholecystic disease among hospital populations of over 30,000 (T.G.H.) and 90,000 (L.A.C.G.H.). This variation is due in part, of course, to the greater number of elective cases in other clinics.

The histories were examined as to 6 cardinal points: (1) chronic indigestion (i.e., painful digestion), flatulence, and so called "bilious attacks" with headaches, as elicited in the past history; (2) dull pain in upper abdomen with radiation posteriorly; (3) well defined biliary colic; (4) presence of jaundice; (5) chills; (6) fever. The relative frequency of these findings for the two series of cases is presented in Tables I and II. In Table III is shown the percentage incidence of these signs and symptoms in the combined series.

Certain points are of interest even in this small series. Jaundice is the one near constant sign; yet one woman with a 3 year history and as many calculi in the common duct failed to show an icteroid tinge at any time, although the icteric index was 13.7. Next in order is a high percentage with history of digestive upset, or chronic upper abdominal pain, or both, over a period of years, or at least months. *There was frequently acute exacerbation of such symptoms when the calculi presumably entered the biliary ducts*, as shown by 81 per cent of the quoted cases. Much of this is due, of course, to activation of chronic cholecystic disease, but a perusal of these clinical histories leads one to believe that any individual who suffers, in mid or late life, a sudden up-trend in severity of previous cholecystic symptoms is an eligible candidate for the diagnosis of common duct stone. If this be associated with icterus and its accompanying manifestations, or failing that an elevated blood bilirubin, or with a dull, radiating, upper abdominal pain, or with chills or fever, the diagnosis becomes more than probable.

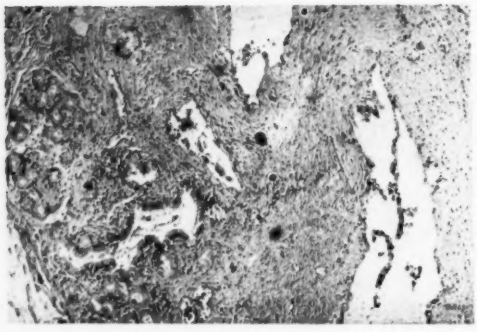

Fig. 4. Low power view of an area from the mid portion of the common bile duct. Virtual absence of muscle fibers. Large groups of hyperplastic glands in duct wall, some of which are cystic and dilated. Hæmatoxylin-eosin stain.

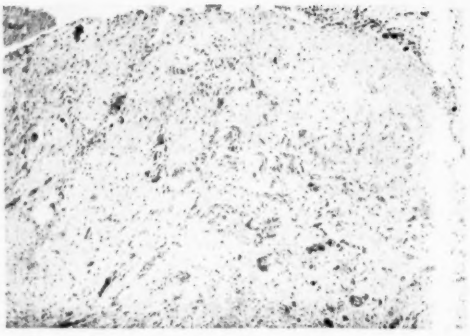

Fig. 5. Low power photomicrograph of an area from the mid portion of a cystic duct. There is complete absence of muscle fibers. Phosphotungstic acid stain.

Most unusual in this series is the extraordinarily low incidence of biliary colic. With the appearance of colic the difficulties of diagnosis are largely erased, but 56 per cent of these cases failed to present such conclusive evidence of common duct stone. In none of the 8 autopsy cases in which the pathologist was the final arbiter was the symptom of colic found in the medical history. This is certain indication of the reluctance with which a diagnosis of biliary duct calculus is surveyed without the *sine qua non* of colic.

Chills were a feature in 7 instances (26 per cent), and fever in 8 (29 per cent).

It should be mentioned that the dull upper abdominal pain complained of by some of these patients is by no means confined to the right upper quadrant. At least 4 (14 per cent) reported left upper quadrant pain, and 3 epigastric pain. Two of the former patients had their radiation of pain to the left scapula.

Only 1 of the 27 cases presented all of the 6 cardinal features herein tabulated, and a second 5. Ten, or 37 per cent, had 4 out of the 6 signs or symptoms, and 6 (22 per cent) only 3. Seven others had only jaundice and one other point, while 1 case presented only jaundice. In the aged, common duct stone, like acute appendicitis, seems remarkable for the lack of clinical evidence produced. Especially is this true of the colic symptom, only patient in this series over 60 presenting this evidence.

Clute's advice regarding more frequent opening of the common duct during operative procedures for cholelithiasis seems to be borne out in this series. Nine of the quoted cases had had previous cholecystectomies or cholecystotomies, and several operative reports stated the bile ducts to be without exterior evidence of calculi. In experienced hands the mortality cannot be appreciably raised by the additional technique and time involved. There is certainly adequate compensation, even if convalescence is lengthened by duct drainage, in the knowledge of a certain result. The formation of calculi in the intrahepatic ducts, as described by Judd and Burden, is of great rarity.

Certain cases of this series merit special remark inasmuch as they illustrate the futility of attempting any reasonable explanation of pain production.

M. C., female, 40 years old, suffering severe colic, was found at operation to have the largest solitary stone in the series, 3 by 1.5 centimeters, in the common duct. It has been said that small stones produce most severe colic. In this instance, at least, a large stone produced severe colic.

J. F., aged 57 years, had had a cholecystectomy in 1923, and was admitted in 1928 with jaundice, fever, and chills. He was without pain or colic, and the autopsy showed a calculus of bean size impacted in the ampulla of Vater, and behind it several smaller stones. The ducts were distended to several times normal diameter throughout. In brief, this individual had failed to develop colic or pain in spite of impaction of a stone in the ampulla, the presence of several small stones, and distention of the ducts—all of which conditions have been named at one time or another as causative of colic.

TABLE I.—PROVED COMMON DUCT STONE

Cases at Los Angeles County General Hospital, 1927–1932

Case	Age	Sex	C. I.	Radiation	Colic	Chills	Fever	Jaundice
M. C. 154.408	46	M.	+	±	+	o	+	+
R. C. 147.776	50	F.	+	+	+	o	±	+
W. E. R. 158.184	64	M.	+	o	o	o	o	+
E. M. 09.767	53	F.	+	+	o	+	+	o
E. I. 178.350	44	F.	+	+	+	o	o	+
J. M. 178.049	70	M.	o	o	+	o	o	+
T. B. 5.941	37	F.	+	o	+	o	o	+
I. W. 99.768	44	M.	+	o	+	+	+	+
J. M. 124.712	60	M.	+	o	o	o	o	+
J. H. 36.269	77	F.	+	+	+	o	o	+
M. N. 10.731	53	F.	+	+	o	o	o	o

Key: C. I.—Chronic indigestion, flatulence, etc.
Radiation—Dull pain with radiation.

J. E. was a man of 70 years, who had suffered from dull epigastric pain for 10 years, but never from colic. In hospital he had a moderate increase of the pain with intermittent chills, fever, and jaundice. Autopsy showed a marble sized stone occluding the ampulla of Vater, with several others in the duct. In the liver were multiple abscesses. Again no colic in spite of multiple stones and occlusion.

M. D., a woman 70 years of age, presented increasing jaundice for 5 months. After admission to hospital her empyemic gall bladder was drained. Autopsy proved the common duct to contain 13 stones of various size. All the hepatic ducts were dilated. She had never suffered even dull pain; nausea, vomiting, and headaches were the only symptoms. Here multiple stones and dilatation of even intrahepatic ducts failed to produce pain.

F. C. was a man of 70 years, whom autopsy showed to have a carcinoma of the gall bladder with complete occlusion of the cystic duct by carcinomatous invasion. He died with a deep jaundice of 5 months' standing which was found at autopsy to be due to stone obstructing the common duct, itself quite patent, and with the remainder of the biliary tree free from carcinomatous invasion. This stone must therefore have been present for many weeks in the common duct (i.e., before the cystic duct became occluded by carcinoma), and was probably responsible for the marked jaundice. His only pain had been epigastric, and of very moderate severity, generally following meals. Thus, in spite of a calculus obstructing the common duct for many weeks—probably 5 months—this individual had never exhibited anything approaching a colic-like pain.

Illustrating the necessity of common duct exploration in doubtful instances is the case of W. E. R., a 64 year old male.

TABLE II.—PROVED COMMON DUCT STONE

Cases at Toronto General Hospital, 1926–1931

Case	Age	Sex	C. I.	Radiation	Colic	Chills	Fever	Jaundice
E. K. 03500	66	F.	+	+	+	o	o	+
J. M. 011673	53	M.	+	+	+	o	o	+
E. C. 019147	22	F.	+	+	+	o	o	+
I. M. 017724	50	F.	+	o	o	o	o	+
F. B. 027624	55	M.	+	o	+	o	o	+
M. C. 024451	40	F.	o	+	+	o	+	+
G. M. 045294	55	M.	+	+	o	o	o	+
K. M. 027085	33	F.	+	+	+	+	+	+
M. C. 040081	65	F.	o	o	o	o	o	+
L. K. 049677	57	F.	o	+	o	o	+	+
J. F. 023273	57	M.	o	o	o	+	+	+
J. E. 021808	70	M.	+	o	o	+	+	+
M. D. 010344	70	F.	o	o	o	+	+	+
H. R. 013608	50	M.	o	o	+	o	o	+
F. C. 04798	70	M.	+	o	o	o	o	+
M. T. 057871	71	F.	+	o	o	o	o	+

TABLE III

	Cases	Per cent
Total cases of common duct stone	27	
Female	15	55
Male	12	45
Average age	56	
Chronic indigestion, flatulence, etc	22	81
Dull pain with radiation	12	44
Biliary colic	12	44
Jaundice	25	92
Chills	7	26
Fever	8	29

Ten years before the last admission a cholecystectomy had been performed, and this was *followed by* jaundice. During the following years he suffered from recurring indigestion and occasional dull epigastric pain. Before admission he developed a constant dull epigastric pain, and pain in the right upper quadrant without radiation, followed by a gradually developing jaundice. In hospital, a preoperative diagnosis of catarrhal jaundice or "adhesions" was made, and the occasional chills he complained of laid to "hysteria." The jaundice cleared, the abdomen was opened, and gastro-enterostomy was done because of dense, distorting adhesions about the pylorus. The biliary ducts were not explored. After a postoperative death, an obstructing calculus was found at autopsy in the common bile duct.

The same point is illustrated by another case:

E. M., a 53 year old woman who 3 years before admission had been operated upon for empyema of the gall bladder. At that time a number of stones were

removed from the gall bladder and the gall bladder drained. She continued to have chronic right upper quadrant pain and flatulence for the 3 years, but no colic at any time. Eventually the common duct was explored and found to contain 3 large stones, and the duct itself was much thickened and dilated.

I. W., a male aged 70 years, illustrates the case with atypical distribution of pain. Having had a cholecystectomy 13 years previously, his chief complaint on admission was left upper quadrant pain radiating to the left scapula. As he presented a marked jaundice and had constant chills and fever, the common duct was explored and a solitary stone ¾ centimeter in diameter removed.

SUMMARY

1. Material has been examined to show that the biliary ducts possess throughout only small isolated fibers of muscle, irregularly scattered. The bile ducts are fibro-elastic tubes, richly supplied with nerve fibers, lined by tall columnar epithelium, and frequently featured by the presence of large subepithelial glands lying in the membrana propria, singly and in groups.

2. Biliary colic is not caused by muscular spasm, according to these histological findings. Several quoted cases go to show that most of the other theories usually advanced as etiological factors of pain are probably false.

3. Colic is by no means a constant symptom of calculi in the bile passages, as shown by an absence of that symptom in 56 per cent of 27 cases presented.

4. The most frequent symptom of common duct stone is an exacerbation of previous indigestion, flatulence, or dull upper abdominal pain with or without radiation. The one near-constant finding is jaundice.

5. A survey of this series impresses one with the need of more frequent exploration of the common duct as a combined procedure with cholecystectomy for cholelithiasis.

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THE BLEEDING TENDENCY IN JAUNDICE[1]

A. C. IVY, M.D., PH.D., P. F. SHAPIRO, M.D., AND P. MELNICK, M.D., CHICAGO, ILLINOIS
From the Departments of Physiology and Surgery of Northwestern University Medical School and the Departments of Surgery and Pathology of the Cook County Hospital

THE surgical and medical risks of infections and other pathological processes in and about the liver are seriously augmented by the possibility of hepatic insufficiency. One of the most striking surgical features associated with these cases of liver damage is their tendency to bleed. According to the results obtained in several clinics, 50 per cent of all postoperative deaths in patients with jaundice or liver insufficiency are a result of hæmorrhage (8, 25, 26, 36). The following study was undertaken chiefly in an effort to reduce these operative risks.

DETERMINATION OF BLEEDING TENDENCY

The first objective was to devise a reliable clinical method that would disclose the latent hæmorrhagic tendency in jaundiced patients. The great variability of bleeding tendency in jaundice is well known. Jaundiced patients with a normal Duke's bleeding time, normal coagulation time, and normal blood coagulating factors will frequently bleed abnormally, and patients with abnormal findings will frequently not bleed. Many clinical and laboratory methods have been recommended to determine the existence of a bleeding tendency, but none of them so far has been an accurate guide. As Colbeck has stated, there is no definite method as yet for the evaluation of the bleeding tendency in jaundice.

1. *Blood coagulation factors. In vitro* studies of the various blood coagulation factors in jaundice have been extensive. Analysis of the literature forces the conclusion that although there are variations in these factors, these variations cannot be correlated reliably with the bleeding tendency. Changes in blood calcium, fibrinogen, platelets, prothrombin, antithrombin, sedimentation rate, etc., are observed just as frequently without as with a hæmorrhagic diathesis in jaundice.

a. *Calcium.* There is undoubtedly a disturbed calcium metabolism in jaundice, such as altered calcium balance, osteoporosis, etc. As one of us (A. C. I., 14) has already pointed out, the literature does not agree on the relation between this disturbance of calcium and the hæmorrhagic diathesis found in some cases. Blood calcium levels, both fixed and diffusible, are unchanged or only slightly changed in jaundice. The general weight of experimental evidence seems to uphold the view that although a functional deficiency of calcium exists, it has no relation to the bleeding tendency (Linton, 22; Snell and Greene; Gunther and Greenberg; Ivy; Wangensteen; Colbeck).

It is true that in jaundice intravenous calcium improves coagulation *in vitro*, but the mechanism by which it does so is not clear. Furthermore, extravascular clotting has little relation to bleeding tendency. Following the lead of Mayo-Robson, Wright and Paramore, Lee and Vincent, Walters (34), Whipple, Judd, and others, the pre-operative administration of calcium is widely used (Cantarow, McKnight, Webb, Brennan, Hanlon, Sistrunk, and others). But failures are frequent. As Wangensteen and also Colbeck have summed up, intravenous calcium has not solved the problem of hæmorrhage in jaundice.

Blood calcium determinations, therefore, are of no value in determining the bleeding tendency. The problem of diffusible calcium presented itself, however, and was studied in many of our cases. The results will be set forth in a separate communication.

b. *Other blood coagulation factors.* Bancroft, Kugelmass and Stanley-Brown, in a study of 200 surgical cases, concluded that no one factor can be isolated as a cause of bleeding or clotting. They devised a "clotting index" calculated from a composite of the determinations of the prothrombin content, fibrinogen content, platelet count, degree of platelet lysis, and antithrombin. In later studies, they dropped the platelet count and lysis studies, because these could not be correlated with clinical bleeding. Another inter-

[1] This work was aided by a grant from Mead Johnson and Company.

esting conclusion was that the clotting tendency can be determined reliably after operation, but not before, because when a latent bleeding tendency exists, the ether damage to the liver and operative shock make it manifest.

For clinical use such a clotting index is entirely too elaborate (Mills, and Clute and Veal). But of greater importance, it is probably inaccurate. The attempt to study the bleeding tendency by splitting it into many factors is misleading. One factor after another is heralded as the important one, only to be dropped when found to be clinically unreliable. For in any given case one factor may compensate for another, both in the calculation and clinically. In different bleeding conditions, different factors are involved. Furthermore, when all factors are normal the patient may still bleed because he may be on the threshold of bleeding and ether, acidosis, operative shock, etc., may push him over this threshold. *It seems as if the only way at present to determine a bleeding tendency is to see if the patient bleeds.* Yet, such *a posteriori* determinations would be of no value in preparing patients for operation.

With particular reference to jaundice, the same conclusions apply. Lewisohn found a decrease in prothrombin and fibrinogen and an increase in antiprothrombin associated with increased risk of hæmorrhage; Murakami and Yamaguchi found a decrease in fibrin ferment in jaundice; Johnson concluded that in experimental obstructive jaundice clotting was delayed because the quantity and quality of the fibrin seems deficient. But Linton found that in obstructive jaundice there is no deficiency in the blood fibrin, in fact a distinct increase. Moss also found a distinct increase in plasma fibrinogen in experimental obstructive jaundice. Mann and Bollman found after removal of the liver that prothrombin did not change, antithrombin sometimes increased and sometimes decreased, and fibrinogen remained normal for a time, but after depletion did not regenerate. The sedimentation rate has also been suggested as a guide to the bleeding tendency in jaundice. Linton (21) considers it to be an accurate guide. Clute and Veal, however, consider it suggestive and helpful, but not absolute.

As with calcium, after much study, determinations of other blood clotting factors were discarded in the present study as not furnishing a reliable guide.

2. *Coagulation time.* Determination of coagulation time is today a most widely used procedure. Yet it seems to have relatively little value. In many hundreds of cases studied by many authors (Kleinert, Hunt, Harter, Feinberg, Bland and Goldstein, Kugelmass, Bancroft and Stanley-Brown, and others) the general conclusions are that prolonged clotting time seems to bear no relation to clinical bleeding, and it is often normal in severe blood dyscrasias and when bleeding occurs. The only condition in which a prolonged coagulation time is diagnostic is hæmophilia. The multiplicity of methods devised for the determination of clotting time points to the difficulty in their evaluation. No less than 33 different methods (probably more) are known (Solis-Cohen). Moreover, the numerous factors affecting the clotting time of shed blood, such as contact with air, glass, and dirt, size and shape of drop, amount of blood, mechanical disturbance of blood, evaporation, temperature, dilution, end point adopted, personal equation, part punctured, contact with tissues, skin and lymph, depth and character of the wound, pressure, meteorological factors, age, hæmoglobin, food, fluid, etc., make the interpretation very difficult. In an exhaustive study of the subject Solis-Cohen has analyzed all these points carefully.

In spite of the unreliability of the method, it was decided in the present study to investigate the coagulation time. The methods of Bogg, Biffis, Lee and White, and Howell were used. Those of Bogg and Biffi were first done on 115 normal individuals to get a standard. Up to 6 minutes was found normal with the Bogg's method (average time of 3 to 5 minutes) and up to 9 minutes for the Biffi. In 60 cases of jaundice, and in 25 miscellaneous pathological cases, the coagulation time was invariably found to be normal. Lee and White's method was done on 32 cases, and Howell's method on 30 cases. These were all normal.

In only 1 jaundice case was the Boggs method above normal, 13 minutes. This patient

was operated upon and died, but of cholæmia, not of hæmorrhage. This would seem to indicate that *the prolonged coagulation time in jaundice is not so much an index of bleeding tendency as of liver damage.* Walters (35) agrees in this view.

Coagulation time, then, was also discarded along with calcium and other determinations as not being an accurate guide.

3. *Bleeding time.* In bleeding conditions, the simple fact is that the patient bleeds. The problem is how to demonstrate beforehand that he is going to bleed. Duke's bleeding time is used universally. To a large extent in jaundice it has been disappointing. Here it is of little more value than the coagulation time. True, in severe anæmia, febrile states, acute leucæmias, and in pronounced hæmorrhagic diatheses in general, it is prolonged, and is the single most reliable method of predicting a bleeding tendency. But a latent bleeding tendency, such as is found in many cases of jaundice, is not revealed by the ordinary puncture of the skin. Thus, it was felt that some way should be found to make a patient with a hypothetical latent bleeding tendency in jaundice bleed excessively from an ordinary skin puncture, and thus possibly make it manifest.

A factor which has received little attention in bleeding studies is the state of "tonicity" of the capillaries. In small vessels it is the retraction of their walls to a large extent which stops bleeding. No other factor can explain the fact that the normal bleeding time of 30 to 180 seconds (Duke) is much less than the normal coagulation time. Von Bermuth and Magnus have demonstrated this clearly, by actual observations of the capillaries with the capillary microscope. Magnus has shown that the normal capillary, when cut, will contract, force out its content of blood, and then disappear completely from view because of its contraction. In a case of hæmophilia, von Bermuth found that the capillaries did not contract after being cut, but remained patent and continued to bleed. In a case of Werlhof's disease, however, the capillaries reacted in the normal manner. This field offers fascinating possibilities for study, and should be investigated further.

It was felt that if the factor of capillary tonus could be eliminated and the capillaries kept wide open, the situation would be simplified and a latent bleeding tendency revealed if present. Such a situation is comparable clinically when ether, shock, operative trauma, etc., results in capillary paresis and bleeding.

The skin of the forearm near the elbow over the pronator muscles was selected as being uniformly thin without the disturbing factor of differences in hornification present on the finger tips. Ordinary Duke's punctures (with mechanical stylet set a uniform depth of about 2.5 millimeters for all cases) gave the usual bleeding time as 30 seconds to 3 minutes. *Heat* was then applied and another puncture done. No constant change was noted. *Cold* was then used; no dependable difference could be noted. *Histamine* (which does not alter blood coagulability, Best and McHenry) was then injected intradermally to produce capillary paresis. No constant changes were found. Altogether 80 cases were studied in this manner.

One of us (A. C. I.) then suggested applying the cuff of a sphygmomanometer around the arm, with a pressure of about 40 millimeters of mercury, enough to cut off effectively the venous return. By increasing the pressure in the capillaries and arterioles in this manner, the factor of "capillary tonus" might be eliminated. This was done, with most gratifying results.

It was found in a large number of normal individuals (115 cases) that very little difference existed between the Duke method and the modified method with venous pressure. In general, the latter gave somewhat longer readings and an increased volume of bleeding, as demonstrated by the total area of the drops on the filter paper. After many trials the upper limit of normal for the venous pressure bleeding time was fixed at 240 seconds; it was rarely over 180 seconds. *But, when the method was tried on a number of cases of jaundice, it was found that often, when the Duke's bleeding time was normal, the venous pressure bleeding time was definitely prolonged.* These cases with a prolonged venous pressure bleeding time were almost always found to be the cases that bled, either spontaneously or after

operation. In the following study of 810 pathological cases, this fact was demonstrated repeatedly. We are not prepared to say just what is the mechanism involved (blood chemical studies of diffusible calcium were made and will be reported separately), but we do feel convinced that the method is of distinct value in predicting a hæmorrhagic diathesis in jaundice.

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THE EFFECT OF VIOSTEROL IN JAUNDICE[1]

R. W. McNEALY, M.D., F.A.C.S., P. F. SHAPIRO, M.D., AND P. MELNICK, M.D., CHICAGO, ILLINOIS

From the Departments of Surgery and Physiology, Northwestern University Medical School and the Departments of Surgery and Pathology of the Cook County Hospital

THE bleeding tendency in jaundice is undoubtedly related to the degree of liver damage. The mechanism of this relation is obscure, but the clinical facts are obvious. The venous pressure bleeding time, or "Ivy" bleeding time (as we have named it) is, therefore, in a sense, a measure of the degree of liver damage.[2] That this is true is also brought out by a study of other types of liver damage without jaundice. The Ivy bleeding time is prolonged in many cases of cirrhosis of the liver, in cases of chronic cholecystitis with associated liver damage, etc. Cases with liver injury, then, formed the bulk of those selected for study.

Dr. R. H. Jaffe suggested to us that a possible explanation for the bleeding tendency in cases of obstructive jaundice was that because of the deficiency of bile in the gastro-intestinal tract, there was a failure in absorption of fat soluble vitamins, particularly vitamin D. As this work was being completed, a publication appeared from the University of California which confirmed this suggestion. Greaves and Schmidt proved that in the absence of bile from the intestinal tract, little or no vitamin D is absorbed. In non-obstructive jaundices or in any liver deficiency with a marked decrease in biliary intestinal content, the vitamin D absorption is not completely inhibited but is definitely impaired. The exhibition of large doses of viosterol or the administration of bile salts with it, we thought might correct this deficiency.

The liver stores vitamin D, and apparently utilizes it in its hæmostatic functions (1, 4). Many reports have been recently published which demonstrate the profound effect of vitamins on the various blood coagulating factors (Kugelmass and Samuel, 9; Woehlisch; Sherif and Baum; Lopez; Cramer and Drew; and others). In particular, the effect of vitamin D in favorably altering these factors and in reducing the bleeding and coagulation time and bleeding tendency in various conditions, has been frequently reported (Phillips, Robertson, Corson and Irwin, 12; Selye; Sanford, Gasteyer and Wyat, 15; Brougher; Richardson; Du Plessis; Kugelmass, 8; Sincock; Shelling; Kaminsky and Davidson; Hess, Lewis and Rirkin, 5; Jones, Rapaport and Hodes, 6; Massengale and Nussmeier, 11; Rohmer and Maryssael, 14; and others). The effect of vitamin D in increasing the calcium content of the blood has received particular attention, and the relationship between blood calcium and blood coagulation is widely accepted. Enteral exhibition of calcium, however, has only a slight effect on blood calcium and parenteral administration has only a transitory effect. Viosterol, it was thought, by its indirect action on calcium metabolism would have a more prolonged and decisive effect in favorably influencing the blood coagulation and bleeding time.

A review of the literature revealed, however, that the evidence for the relationship between blood calcium and blood coagulation, and for the relationship between vitamin D and blood calcium was in many cases indecisive and contrary. We decided, therefore, to study in our own cases the relationship between viosterol administration and blood calcium (this work will be reported in a separate paper) and in the meantime to determine clinically whether viosterol actually would decrease the bleeding tendency of cases of jaundice or hepatic insufficiency.

Eight hundred and ten unselected cases at the Cook County Hospital were studied in a period of 14 months. Almost all the jaundiced patients who entered the hospital during this period were studied, namely, those with common duct stone, cirrhosis of the liver with jaundice, toxic hepatitis catarrhal jaundice, primary and metastatic carcinoma of the liver,

[2] See Ivy, A. C., Shapiro, P. F., and Melnick, P. The bleeding tendency in jaundice. Surg., Gynec. & Obst., 1935, 60: 781-784.

[1] This work was aided by a grant from Mead Johnson and Company.

carcinoma of the extrahepatic bile ducts and of the head of the pancreas, etc. Another large group included patients with liver damage but without jaundice, such as cirrhosis of the liver without jaundice, chronic cholecystitis, and cholelithiasis, etc. A miscellaneous group of jaundiced patients included pneumonia with jaundice, cardiac decompensation with jaundice, etc. In addition, various types of miscellaneous patients were studied, mainly as controls, such as various types of blood dyscrasias and anæmias, purpuras, tonsil cases with bleeding history, etc.

The patients were grouped broadly into surgical and medical patients. In the surgical patients the practical aspect of postoperative bleeding was a measure of the effect of viosterol, in addition to the lowered Ivy bleeding time. In the medical cases, the Ivy bleeding time was of chief value, although cessation of spontaneous bleeding from the gastro-intestinal tract or other sources was also an indication of viosterol effect. In general, the average dose of viosterol was 30 drops of 250D three times a day.[1] *In cases with acholic stools, bile salts were also administered to insure the absorption of vitamin D.* In addition, calcium and glucose were sometimes administered orally or intravenously. This administration was irregular and was done in the control cases in about the same proportion as in the definitive group. For a time, viosterol 10,000X was given to some patients, but no difference could be noted in its action and it was discontinued. In general, where the bleeding time was not prolonged, no viosterol was given; the course was usually uneventful. About one-third of the patients with a prolonged bleeding time, unselected, were used as controls and were not given viosterol.

Altogether, 810 patients were studied. There were 376 surgical patients and 434 medical patients.

SURGICAL CASES

1. Chronic cholecystitis and cholelithiasis. Among the surgical patients 254 were patients with chronic cholecystitis and cholelithiasis, upon whom a cholecystectomy was performed. In this group there were 219 (19 colored and 200 white) who showed no bleeding tendency in any of the preliminary tests. They included 30 men and 189 women. No viosterol was given. In the whole group there were only 3 deaths, all from extrahepatic complications. One patient died of cardiac decompensation 2 weeks after operation, and 2 of bronchopneumonia 10 days after. One patient ran a stormy postoperative course. The rest, 215 patients, made an uneventful recovery. The Ivy bleeding time thus seems to have good prognostic value, for when it is normal, good results can be expected, and viosterol is not indicated.

Thirty-five, or 13.7 per cent, of the patients (3 men and 32 women) in this group (Table I) showed a bleeding tendency on the preliminary tests. The Duke bleeding time averaged 120 seconds, well within the normal range, but the Ivy bleeding time averaged 320 seconds, quite above the normal upper limit of 240 seconds. Nineteen of the 35 patients gave a history of abnormal bleeding. The icteric index averaged 11. Of these 35 patients with a bleeding tendency, 24 were given viosterol for from 4 days to 2 weeks before operation, in doses of 30 to 60 drops, three times a day. After this therapy, the Duke bleeding time averaged 80 seconds, the Ivy bleeding time dropped to 130 seconds. Twenty-two of these patients made uneventful postoperative recoveries and two ran stormy postoperative courses. There were no deaths.

As a control series, 11 of the patients were not given viosterol. Without viosterol preparation, the Duke bleeding time before operation averaged 132 seconds, and the Ivy bleeding time averaged 346 seconds, still well above normal.

Thus, in these cases there was no spontaneous improvement in the bleeding time. One patient died in cholæmia, and 10 ran stormy postoperative courses, requiring blood transfusions and 4 to 6 weeks' stay in the hospital. There were no smooth recoveries. Here too the Ivy bleeding time was seen to have prognostic value. When it is increased, trouble can be expected unless it is first reduced by viosterol therapy.

It is significant that at operation no unusual difficulty with hæmostasis was noted, even in the control cases. The patients "went bad"

[1] The viosterol and other drugs used in this work were kindly supplied by Mead Johnson and Company.

TABLE I.—SURGICAL GALL-BLADDER CASES WITH BLEEDING TENDENCY

Case number	Sex and color	Age	Icteric index	Bleeding history	Duke bleeding time	Ivy bleeding time	Viosterol	After viosterol					Results
								Bleeding	Duke bleeding time	Ivy bleeding time	Operation	Postoperative bleeding	
153	♂	42	12	o	110	290	30 drops t.i.d. 1 wk.	o	60	150	+	o	Recovered
155	♀	48	5	o	140	310	30 drops t.i.d. 1 wk.	o	70	110	+	o	Recovered
156	♀	42	19	±	130	310	30 drops t.i.d. 1 wk.	o	50	90	+	o	Recovered
447	♀	43	8	±	150	360	30 drops t.i.d. 1 wk.	o	60	120	+	o	Recovered
448	♀	27		o	160	290	30 drops t.i.d. 1 wk.	o	60	100	+	o	Recovered
449	♀	40		o	140	290	30 drops t.i.d. 1 wk.	o	70	120	+	o	Recovered
63	♀	33	5	++	100	440	30 drops t.i.d. 1 wk.	o	40	150	+	o	Recovered
226	♀	37	18	+	160	390	30 drops t.i.d. 1 wk.	o	70	150	+	o	Stormy convalescence; recovery
227	♀	31	12	±	180	330	60 drops t.i.d. 1 wk.	o	40	100	+	o	Recovered
228	♀	42	4	o	100	280	30 drops t.i.d.	o	70	110	+	o	Recovered
229	♀	46	5	±	190	320	30 drops t.i.d.	o	70	100	+	o	Recovered
230	♀	43	20	o	180	450	60 drops t.i.d. 1 wk. 60 drops t.i.d. 2 wks.	o	150 100	400 140	+	o	Stormy convalescence
231	♀	41	16	o	110	260	30 drops t.i.d.	o	60	130	+	o	Recovery normal
510	♀	27		+	180	420	30 drops t.i.d.	o	70	150	+	o	Recovery normal
511	♀	40		++	120	290	30 drops t.i.d.	o	90	150	+	o	Recovery normal
512	♀	63	6	+	90	400	30 drops t.i.d.	o	60	180	+	o	Recovery normal
513	♀	24		o	100	300	30 drops t.i.d.	o	50	140	+	o	Recovery normal
583	♀	58		o	80	280	20 drops t.i.d.	o	60	110	+	o	Recovery normal
584	♀	44	5	o	90	290	20 drops t.i.d.	o	60	150	+	o	Recovery normal
585	♀	64	12	+	100	270	20 drops t.i.d.	o	50	110	+	o	Recovery normal
586	♀	44		+	140	280	20 drops t.i.d.	o	80	120	+	o	Recovery normal
587	♀	54	8	o	90	280	30 drops t.i.d.	o	50	120	+	o	Recovery normal
752	♀	24		+	100	290	30 drops t.i.d.	o	40	50	+	o	Recovery normal
588	♀	53		o	70	260	30 drops t.i.d.	o	50	70	+	o	Recovery normal
361	♀	46	20	±	110	360	None	+	140	340	+	+	Stormy P.O. course; transfusion C.C.H. 4 wks.
653	♀	36		o	90	320	None	o	120	300	+	o	Stormy P.O. course; transfusion C.C.H. 4 wks.
654	♀	56		o	90	270	None	o	90	330	+	o	Stormy P.O. course; transfusion C.C.H. 4 wks.
655	♀	33	24	+	110	300	None	o	130	360	+	+	Stormy P.O. course
656	♀	65	8	+	80	290	None	o	100	360	+	o	Stormy P.O. course
657	♀	38		+	190	400	None	+	160	400	+	+	Stormy P.O. course; transfusion
154	♀	50	15	o	60	290	None				+	+	Died in 3 days
450	♂	43		±	190	290	None 1 wk. P.O. 3 wks. P.O. 4 wks. P.O.	± + o o	190 150 110 90	380 340 250 210	+	+	Stormy P.O. course; recovered after 4½ wks.
311	♀	44	20	±	90	290	None 1 wk. P.O. 2 wks. P.O. 3 wks. P.O.	o o o o	130 70 80 60	300 270 170 100	+	±	Collapse 3 hrs. P.O.; blood transfusion 4 wks. P.O. ad; recovery
312	♂	39	38	+	150	330	None 1 wk. P.O. 2 wks. P.O. 3 wks. P.O.	o o o	90 80 60	240 160 110	+	±	Collapse P.O.; transfusion 4 wks. ad; recovery
753	♀	42		+	150	320	None	±	110	300	+	+	Stormy convalescence course; transfusion

8 to 24 hours after operation. It occurred to us here that the reason for this is that jaundiced patients clot as quickly as normals, but that their clots are fragile, and the postoperative retching, etc., breaks them loose.

We have spent a great deal of time in studying the clots of these patients. The increased fragility of their clots in comparison with normal clots is obvious, but we have as yet been unable to devise an instrument to record this fragility accurately.

2. Common bile duct stone. Sixty-four patients with common bile duct stone were operated upon. Of these, 27, 8 men and 19 women, showed no bleeding tendency. Their icteric index ranged from 16 to 150; their ages from 24 to 68. None of the 27 received viosterol except patient 26. All made uneventful postoperative recoveries except patients 26 and 24. Therefore in patients with normal Ivy bleeding time, smooth spontaneous recovery can usually be expected without viosterol. Patient 24 was a white woman 46 years old. She had an icteric index of 35, no bleeding history, and a bleeding time which was just within normal limits. She was in excellent general condition. She was not given viosterol; but the night before operation was given 5 cubic centimeters of 10 per cent calcium chloride intravenously. She died suddenly 1 hour after the injection. Patient 26 was a white man 58 years old. He had an icteric index of 130. There was no bleeding history. He had a normal bleeding time, but was in poor general condition. For this reason he was given 30 drops of viosterol three times a day for 10 days. He died 4 days after operation in cholæmia.

Thirty-seven, or 57 per cent, of the patients with common duct stone (11 men and 26 women) had an abnormally prolonged bleeding time. The Duke bleeding time averaged 125 seconds, but the Ivy bleeding time averaged 330 seconds. The icteric index averaged 58. Twenty-four of the 37 patients were given viosterol. The bleeding tendency stopped in all, except patient 762. The Duke's bleeding time dropped in from 1 to 2 weeks to an average of 65 seconds, the Ivy bleeding time to 120. There was only 1 death, patient 762, and only 1 stormy postoperative course.

As a control series, 13 of the 37 patients were not given viosterol. The bleeding tendency persisted in 11 of these 13 patients, the Duke's bleeding time rose to an average of 140 seconds, the Ivy bleeding time rose to an average of 385. There were 5 deaths, 8 stormy postoperative courses, and no uneventful recoveries. This stands in striking contrast to the group with prolonged bleeding time who were given viosterol (Table II).

3. Surgical malignancies. Twenty-four surgical patients with carcinoma of the liver or of the extrahepatic bile ducts were studied. There were 16 men and 8 women. Fourteen of these patients (10 men and 4 women) showed no bleeding tendency. Their ages varied from 40 to 72. Their icteric index varied from 22 to 120. No viosterol was given. Thirteen of the fourteen had exploratory laparotomies. One patient was in very poor condition when operated upon and died a few hours later. The rest made uneventful postoperative recoveries. One patient had a cholecystogastrostomy with an uneventful recovery.

Ten, or 41 per cent, of the patients (6 men and 4 women) showed a bleeding tendency (Table III). Six gave a history of bleeding. The icteric index averaged 56. The Duke bleeding time averaged 120 seconds, the Ivy bleeding time 430 seconds. Eight of the 10 patients were given viosterol. The Duke's bleeding time within from 5 to 12 days dropped to an average of 75, the Ivy bleeding time to 190 seconds. Two patients on viosterol retained their bleeding tendency. One died in cholæmia. The other ran a stormy postoperative course. A third patient was relieved of his bleeding tendency, but died of cardiac decompensation 2½ weeks after operation. Five of the 6 whose bleeding tendencies were reduced made uneventful postoperative recoveries. As a control series, 2 patients were not given viosterol. The bleeding time remained unchanged. Both died in cholæmia.

In these patients, too, the same general relationship holds between the bleeding time and the prognosis. Also, the effect of viosterol in keeping down the bleeding time and bleeding tendency for a time was noted. However, where the liver is hopelessly damaged and rid-

TABLE II.—SURGICAL COMMON BILE DUCT CASES WITH BLEEDING TENDENCY

Case number	Sex and color	Age	Icteric index	Bleeding history	Duke bleeding time	Ivy bleeding time	Viosterol	After viosterol					Results
								Bleeding	Duke bleeding time	Ivy bleeding time	Operation	Postoperative bleeding	
234	♀	45	88	+	120	310	30 drops t.i.d. 2 wks.	o	50	80	+	o	Recovered
235	♀	38	58	±	160	320	30 drops t.i.d. 2 wks.	o	50	90	+	o	Recovered
236	♀	48	21	o	100	260	60 drops t.i.d. 1 wk.	o	70	130	+	o	Recovered
237	♀	48	65	o	120	250	30 drops t.i.d. 1 wk.	o	50	90	+	o	Recovered
238	♂	40	62	+	130	270	30 drops t.i.d. 3 days	o	50	100	+	o	Recovered
239	♀	42	22	±	110	290	30 drops t.i.d. 1 wk.	o	50	80	+	o	Recovered
161	♂	62	36	o	70	290	30 drops t.i.d. 1 wk.	o	60	130	+	o	Stormy P.O. course
452	♀	38	44	o	140	300	30 drops t.i.d. 1 wk.	o	80	120	+	o	Recovered
453	♀	37	50	o	140	290	30 drops t.i.d. 5 d.	o	80	120	+	o	Recovered
454	♀	23	46	o	280	480	30 drops t.i.d. 2 wks.	o	80	120	+	o	Recovered
87	♀	31	36	+	50	240	60 drops t.i.d. 1 wk.	o	60	130	+	o	Recovered
88	♀	28	89	+	150	240	60 drops t.i.d. 1 wk.	o	30	120	+	o	Recovered
316	♂	46	260	+	180	360	30 drops t.i.d. 10 d. + bile salts 7 gr. t.i.d.	o	80	190	+	o	Recovered
10	♂	38	53	o	50	260	30 drops t.i.d. 10 d.	o	80	180	+	o	Recovered
695	♂	30	50	+	120	380	30 drops t.i.d. 1 wk. + bile salts 7 gr. t.i.d.	o	50	80	+	o	Recovered
696	♂	61	45	+	100	310	30 drops t.i.d. 1 wk. + bile salts 7 gr. t.i.d.	o	50	90	+	o	Recovered
514	♀	44	25	o	80	280	20 drops t.i.d. 1 wk.	o	60	130	+	o	Recovered
515	♀	60	32	+	110	300	20 drops t.i.d. 1 wk.	o	60	150	+	o	Recovered
516	♂	27	38	+	110	510	30 drops t.i.d. 1 wk.	o	100	150	+	o	Recovered
2	♀	27	28	++	160	280	60 drops t.i.d. 1 wk.	o	140	100	+	o	Recovered
5	♂	49	16	++	50	250	30 drops t.i.d. 1 wk.	o	50	150	+	o	Recovered
37	♂	47	34	+	120	300	30 drops t.i.d. 1 wk.	o	60	80	+	o	Recovered
761	♀	50	150	o	90	280	30 drops t.i.d. 1 wk. + bile salts 7 gr. t.i.d.	o	60	180	+	o	Recovered
762	♀	50	77	+	130	400	30 drops t.i.d. 1 wk. + bile salts 7 gr. t.i.d.	o	60	230	+	o	Died
162	♀	36	63	+	160	420	No viosterol	+	140	400	+	+	Died a few hrs. P.O. of hæmorrhage
318	♀	52	82	±	180	410	No viosterol	+	180	450	+	+	Died 1 hr. P.O. of hæmorrhage
697	♀	62	12	+	90	300	No viosterol	+	150	300	+	+	Died 1 day later
698	♀	39	65	+	170	480	No viosterol	+	160	380	+	+	Died 1 day later; no autopsy
699	♀	39	36	o	100	360	No viosterol	o	80	320	+	o	Stormy P.O. course
700	♀	47	16	+	100	420	No viosterol	o	90	380	+	o	Stormy P.O. course
680	♀	42	28	+	80	270	No viosterol	+	90	300	+	+	Stormy P.O. course
384	♀	29	80	+	70	360	No viosterol 2 wks. P.O.	o o	140 120	460 400	+ +	o o	Stormy P.O. course Still in C.C.H.
317	♀	27	220	++	280	500+	No viosterol 2 wks. P.O. 5 wks. P.O.	+ + o	230 260 100	500+ 480 200	+ + +	+	P.O. collapse; transfusion 5 wks.; recovery
758	♂	40	++	o	110	330	No viosterol				+	o	Stormy P.O. course
757	♂	55	+++	+	60	420	No viosterol	+	90	490	+	o	Stormy P.O. course
759	♀	44	38	±	100	330	No viosterol	+	100	380	+	o	Stormy P.O. course
760	♀	51	36	+	70	310	No viosterol				+	+	Died

TABLE III.—SURGICAL MALIGNANCIES WITH BLEEDING TENDENCY AND VIOSTEROL

Case number	Sex and color	Age	Diagnosis	Icteric index	Bleeding	Duke	Ivy	Viosterol	Explanatory Operation				Result
									After viosterol				
									Bleeding	Operation	Duke	Ivy	
166	♂	52	Secondary carcinoma of liver	26	0	100	270	30 drops t.i.d. for 1 wk.	0	+	50	100	Recovered
167	♀	58	Secondary carcinoma of liver	32	0	100	260	30 drops t.i.d. for 1 wk.	0	+	60	130	Recovered
517	♀	39	Secondary carcinoma of liver	65	+	170	480	30 drops t.i.d. for 1 wk.	0	+	60	180	Recovered
518	♂	58	Secondary carcinoma of liver	72	+	150	960	30 drops t.i.d. for 1 wk.	0	+	130	120	Recovered
91	♀	61	Carcinoma of gall bladder	90	++	160	480	30 drops t.i.d. for 1 wk. +calcium lactate, glucose, and transfusion after 2 wks.	+	+ + +	90	260	Died in 24 hrs. in cholæmia (Boggs, 13 min.)
240	♂	54	Carcinoma of gall bladder	50	0	140	260	30 drops t.i.d. 10 days	0	+	70	140	Died in 2½ wks. of cardiac decompensation
3	♂	48	Carcinoma of gall bladder	54	++	70	540	30 drops t.i.d. 1 wk. + calcium gluconate	±	+	60	240	Stormy P.O. course
763	♀	49	Carcinoma papilla of Vater	38	0	60	270	30 drops t.i.d. 1 wk. + bile salts	0	+	50	90	Recovered
319	♂	40	Secondary carcinoma of liver	46	+	160	350	No viosterol	+	+	150	380	Died 1 day P.O.
35	♂	73	Secondary carcinoma of liver	70	0	60	280	No viosterol	0	+	90	280	Died 1 day P.O.

dled by carcinoma, nothing will help it. We were able to follow the gradually advancing destruction of the liver by the gradually increasing bleeding time, and the failure of the patient to respond to viosterol.

4. Miscellaneous surgical cases. There were 2 patients with liver abscess. Neither showed a bleeding tendency. No viosterol was given. They were drained surgically, and made uneventful recoveries. One patient with biliary cirrhosis, patient 393, was operated. She was 33 years old and had an icteric index of 150, and no bleeding tendency. She was not given viosterol. While in the hospital, her Ivy bleeding time increased from 160 to 300. She was explored at that time. The postoperative course was stormy, and the prolonged bleeding time continued for 4 weeks, when she began to recover slowly. One patient with a splenic anæmia was operated upon. She was 55 years old and had a very high bleeding tendency (380 seconds Duke, 460 Ivy). No viosterol was given. Splenectomy was done. The patient died 1 week later with recurrent hæmorrhages. One patient with a thrombocytopenic purpura, patient 764, had a Duke bleeding time of 510 seconds and an Ivy bleeding time of 670, and a platelet count of 70,000. She was put on viosterol, but transfusions were also given. Her bleeding time dropped to normal and the platelet count rose to 150,000. Recovery after splenectomy was uneventful.

During the course of the year, 29 tonsil patients were found (out of about 5,000 operated upon) who gave a bleeding history (15 males and 14 females). Their ages varied from 4 to 31 years. Only one of these, however, showed a slightly prolonged bleeding time (260 Ivy). No viosterol was given to any case. All, even the latter, made uneventful recoveries. There were no cases of serious bleeding other than a few cases on the basis of definite surgical injury to larger vessels. There were no deaths. Because of inadequate "bleeding" material, nothing can be said regarding the use of viosterol before tonsillectomy.

MEDICAL CASES

1. Chronic cholecystitis and cholelithiasis. Eighty-two patients with chronic gall-bladder disease entered the hospital with symptoms of biliary colic, acute gastro-intestinal upsets, or chronic dyspepsia. There were 11 men and 71 women. Of these, only 1 showed a bleeding tendency. No viosterol was given. All recovered promptly from their admission symp-

TABLE IV.—MEDICAL COMMON BILE DUCT CASES

Case number	Sex and color	Age	Icteric index	Bleeding	Duke	Ivy	Viosterol	After viosterol			Result
								Bleeding	Duke	Ivy	
534	♀	26	29	o	90	470	30 drops t.i.d. 2 wks. + bile salts 2 wks.	o	90	150	Improved; refused operation
795	♀	20	19	o	90	410	30 drops t.i.d. 2 wks. + bile salts 2 wks.	o	70	150	Improved; refused operation
704	♂	50	62	+	70	450	30 drops t.i.d. 10 days + bile salts 10 days	o	50	140	Improved; refused operation
676	♂	43	31	o	50	240	Calcium only	o	60	90	Slow improvement; refused operation
795	♀	23		++	160	440	No viosterol	++	200	430	Died 2 days
782	♀	45	38	o	80	390	30 drops t.i.d. 1 wk. + bile salts	o	80	200	Refused operation
781	♂	48	++	o	50	80	No viosterol	±		310	Refused operation

toms. Operation was not advised for the time being in many of the cases; in the remainder, the patients refused operation and left the hospital. One patient, 355, a man 47 years old, entered the hospital with an acute cholecystitis. He had a bleeding history and a prolonged bleeding time (Duke 230 seconds; Ivy 550). He was not given viosterol. Operation was advised but he refused and left the hospital.

2. Common duct stone. Fourteen patients with common bile duct stone were studied who did not come to operation. There were 8 men and 6 women. Seven patients showed no bleeding tendency. They had an icteric index of 27 to 70. No viosterol was given. All made a spontaneous temporary recovery from their jaundice and gastro-intestinal symptoms.

Seven patients manifested a bleeding tendency (Table IV). Two gave a history of bleeding; Duke's bleeding time averaged 90 seconds; Ivy bleeding time, 400 seconds. The icteric index averaged 38. Four of these were given viosterol and bile salts. The Duke bleeding time within 1 to 2 weeks averaged 70; the Ivy bleeding time dropped to 140 seconds. All 4 recovered. One patient received calcium only. She improved slowly. Two patients received no medication. The bleeding time remained unchanged in 1 and the patient died. In the other, it grew steadily longer and the patient steadily worse. The prognostic value of the Ivy bleeding time and efficacy of viosterol in improving prognosis is shown in these as in the surgical patients.

3. Empyema of gall bladder. One patient with empyema of the gall bladder, patient 356, a man 44 years old, was admitted in poor condition. Viosterol was not tried. He died in 2 days. Another similar patient, 601, was prepared with viosterol. She began to recover spontaneously and refused operation. The correct diagnosis in this case is in question.

4. Catarrhal jaundice. Fifty-one patients with catarrhal jaundice were studied, 36 men and 15 women. Thirty-six patients showed no bleeding tendency. They had an icteric index of from 18 to 100. Their ages varied from 15 to 55. No viosterol was given. All recovered promptly within 2 weeks.

Fifteen patients, or 29 per cent, had a prolonged bleeding time; the Duke bleeding time averaged 115 seconds; the Ivy, 330 seconds (Table V). Twelve gave a history of abnormal bleeding. The icteric index averaged 58. Twelve of the 15 were given viosterol. The bleeding tendency stopped in all within 1 to 3 days. The Duke bleeding time dropped within 3 to 6 days to an average of 70 seconds, the Ivy to 100 seconds. All 12 recovered promptly within from 5 days to 2 weeks. Three of the 15 were not given viosterol. One was ill for 7 weeks, 1 for 6 weeks, and only 1 recovered with usual promptness. Although catarrhal jaundice is a self-limited disease, it seems that when a prolonged Ivy bleeding time clouds the prognosis, viosterol is of value.

5. Cirrhosis of the liver. There were 104 patients with cirrhosis, 56 men and 48 women. Most of these were periportal cirrhosis, a few were biliary. Only 4 of the patients were colored.

Forty-seven patients showed no bleeding tendency. No viosterol was given, but the

TABLE V.—CATARRHAL JAUNDICE

Case number	Sex and color	Age	Icteric index	Bleeding history	Duke bleeding time	Ivy bleeding time	Viosterol	After viosterol			Result
								Bleeding	Duke bleeding time	Ivy bleeding time	
94	♀	24	46	+	120	280	30 drops t.i.d. 1 wk.	0	60	140	Recovery 1 wk.
95	♂	31	75	+	70	300	30 drops t.i.d. 10 days	0	80	120	Recovery 10 days
96	♀	28	36	++	120	310	60 drops t.i.d. 1 wk.	0	80	170	Recovery 1 wk.
538	♀	45	96	+	90	270	20 drops t.i.d. 2 wks.	0	50	90	Recovery 2 wks.
1	♂	52	88	+++	160	260	30 drops t.i.d. 10 days	0	80	130	Recovery 10 days
8	♂	39	50	+	80	300	30 drops t.i.d. 2 wks.	0	80	130	Recovery 2 wks.
53	♂	43	35	+++	70	550	30 drops t.i.d. 1 wk.	0	60	90	Recovery 1 wk.
58	♀	14	75	+++	70	400	30 drops t.i.d. 2 wks.	0	70	210	Recovery 2 wks.
457	♂	43	28	0	150	370	30 drops t.i.d. 3 days	0	50	100	Recovery 5 days
256	♂	43	103	++	130	260	30 drops t.i.d. 1 wk.	0	70	130	Recovery 1 wk.
257	♀	33	45	+	160	330	60 drops t.i.d. 1 wk.	0	80	140	Recovery 1 wk.
254	♀	53	22	0	90	260	20 drops t.i.d. 1 wk.	0	60	90	Recovery 1 wk.
332	♀	29	69	+	190	340	No viosterol—7 wks.	0	90	180	Finally recovered after 7 wks.
678	♀	24	63	+	100	380	No viosterol—6 wks.	0	80	350	Left hospital after 6 wks.; condition only fair.
725	♀	26	34	0	120	320	No viosterol—10 days	0	60	90	Recovery 10 days

patients were under observation in the hospital for from 3 to 6 weeks. Their icteric index ranged from 7 to 94, their ages from 36 to 62. Forty-two of these patients were stationary while under observations or improved slightly. One patient, 468, a man 38 years old, had a low bleeding time but was nevertheless moribund on admission. He died the next day.

Fifty-seven patients, or 54 per cent, with cirrhosis had a prolonged bleeding time. The Duke bleeding time averaged 128 seconds, the Ivy 350 seconds. Thirty-eight of the 57 cases gave a history of abnormal bleeding. The icteric index averaged 44.

Thirty of the 57 patients were given viosterol (Table VI). Six of them died in the course of 3 to 6 weeks, although none was moribund on admission. *The bleeding tendency of these 6 did not abate on the administration of viosterol.* The Duke bleeding time rose to 190, the Ivy to 460. Two patients were stationary, one made slight improvement. Four patients improved moderately, and 17 patients improved markedly while under viosterol treatment. In the 24 patients in this group on viosterol who survived, the bleeding tendency subsided in from 1 to 4 weeks. The Duke time fell in from 1 to 4 weeks to an average of 75 seconds, the Ivy to 150 seconds. In 2 of the patients who were improving, the viosterol was stopped. One of them became rapidly worse but is still alive, the other died.

Twenty-seven of the 57 patients with bleeding tendency were not given viosterol despite their prolonged bleeding time (Table VII). There were 14 deaths, but 9 patients were moribund on admission; therefore, only 5 deaths can be counted to compare with the cases receiving viosterol. The striking contrast was that 9 of the control patients ran a progressive downhill course, 1 was stationary, 1 showed a slight improvement, and only 2 showed a marked improvement when viosterol was withheld. In these last 13 patients who are still living, the Duke bleeding time averaged 90 seconds, the Ivy time remained at 310.

It seems that in cirrhosis of the liver with a prolonged bleeding time, if the process is early or slow in its progress, viosterol has some

TABLE VI.—CIRRHOSIS CASES WITH BLEEDING TENDENCY AND VIOSTEROL

Case number	Sex and color	Age	Icteric index	Bleeding history	Duke bleeding time	Ivy bleeding time	Viosterol	After viosterol			Results
								Bleeding	Duke bleeding time	Ivy bleeding time	
247	♂	51	28	0	60	300	30 drops t.i.d. 2 wks. 4 wks.	0 0	80 60	220 110	Marked improvement
607	♀	42	34	+	130	360	30 drops t.i.d. 3 wks.	0	70	170	Marked improvement
715	♀	40	5	0	90	280	30 drops t.i.d. 3 wks.	0	60	100	Marked improvement
717	♂	24	22	+	130	610	30 drops t.i.d. 4 wks.	0	50	210	Marked improvement
718	♂	42		+	50	250	30 drops t.i.d. 3 wks.	0	60	100	Marked improvement
549	♂	24	84	++	130	610	30 drops t.i.d. 2 wks.	0	50	210	Marked improvement
716	♀	48	44	+++	150	380	30 drops t.i.d. 5 wks.	0	70	190	Remarkable recovery. Viosterol stopped in 4 wks. Began to go bad; 2 wks. later, bleeding began again. Duke 120, Ivy 430.
248	♂	38		0	90	210	30 drops t.i.d. 2 wks.	0	60	90	Moderate improvement
100	♂	30	9	+	130	280	60 drops t.i.d. 3 wks.	0	100	160	Moderate improvement
101	♀	50	14	+	90	280	60 drops t.i.d. 3 wks.	0	90	180	Moderate improvement
249	♀	56	26	0	110	260	60 drops t.i.d. 2 wks.	0	80	160	Moderate improvement
251	♂	60	20	0	110	260	30 drops t.i.d. 2 wks.	0	70	130	Moderate improvement
177	♀	36	28	+	120	300	30 drops t.i.d. 2 wks.	0	60	100	Moderate improvement
178	♀	37	20	0	120	270	30 drops t.i.d. 2 wks.	0	70	100	Moderate improvement
61	♀	42	26	+	200	270	30 drops t.i.d. 3 wks.	0	100	120	Moderate improvement
713	♀	40	24	++	110	420	30 drops t.i.d. 4 wks.	0	70	180	Moderate improvement
714	♂	42		+	180	500	30 drops t.i.d. 5 wks.	0	60	150	Moderate improvement
465	♂	64		0	180	900	30 drops t.i.d. 4 wks.	0	180	350	Moderate improvement
466	♀	33	150	++	220	460	30 drops t.i.d. 6 wks.	0	160	340	Moderate improvement
179	♂	26	16	+	160	280	30 drops t.i.d. 3 wks.	0	80	160	Slight improvement
250	♀	56	18	±	140	310	60 drops t.i.d. 4 wks.	0	100	190	Slight improvement
13	♂	38	86	+++	120	360	30 drops t.i.d. 1 wk. 60 drops t.i.d. 2 wks. 90 drops t.i.d. 2 wks.	+++ +++ +++	120 240 230	330 400 300	No improvement No improvement No improvement
32	♀	40	37	+++	140	400	60 drops t.i.d. 2 wks.	++	130	400	Steadily worse; died
33	♀	33	76	+	90	300	60 drops t.i.d. 10 days	+	90	280	Steadily worse; died
27	♀	41	6	+	80	260	30 drops t.i.d. 3 wks.	++	200	610	Steadily worse; died
719	♂	51	25	++	200	330	60 drops t.i.d. 4 wks. 5 drops t.i.d. (10,000X) 4 wks.	+++ +++	230 300	460 500+	Steadily worse; died Steadily worse; died
176	♀	47	73	++	140	450	30 drops t.i.d. 1 wk. 30 drops t.i.d. 1 wk. No viosterol 1 wk. No viosterol 1 day more	+ 0 +	120 60 100	270 70 290	Died of massive hæmatemesis
546	♂	51		++	200	330	30 drops t.i.d. 1 wk. 60 drops t.i.d. 1 wk. 10,000X—5m t.i.d., 1 wk.	++ +++ ++++	250 270 300	420 480 500+	Died
805	♂	45	147	0	390+		30 drops t.i.d. 1 wk.		310	460	No change
806	♀	43	16	0	130	430	30 drops t.i.d. 1 wk.		110	300	Slight improvement

value, while if it is advanced or rapid in its progress viosterol has no demonstrable value.

6. Malignancy. There were 68 patients with malignancy in and about the liver who did not come to operation. They included 41 men and

TABLE VII.—CIRRHOSIS CASES WITH BLEEDING TENDENCY, WITHOUT VIOSTEROL

Case number	Sex and color	Age	Icteric index	Bleeding history	Duke bleeding time	Ivy bleeding time	Viosterol	Bleeding	Duke bleeding time	Ivy bleeding time	Result
465	♂	64		+		310	No viosterol 10 days	o	70	140	Marked improvement
712	♂	48		o	50	550	No viosterol 3 wks.	o	30	110	Marked improvement after an attack of erysipelas
711	♀	43	26	o	160	380	No viosterol 2 wks.	o	140	300	Slight improvement
330	♀	66	8	o	110	260	No viosterol 3 wks.	o	130	300	Stationary
400	♀	59	38	o	80	270	No viosterol 3 wks.	o	120	290	Gradually worse
401	♀	30		o	80	260	No viosterol 4 wks.	o	100	260	Gradually worse
402	♀	48	62	+	100	300	No viosterol 4 wks.	+	110	320	Gradually worse
682	♀	52		+	130	400	No viosterol 4 wks.	+	150	450	Gradually worse
683	♀	51	40	+	100	380	No viosterol 4 wks.	±	90	420	Gradually worse
684	♀	17		o	130	280	No viosterol 4 wks.	±	130	320	Gradually worse
467	♂	36	112	++	200	360	No viosterol 2 wks.	++		580	Rapidly worse
398	♂	52		+	60	530	No viosterol 1 wk.	+		500	Rapidly worse
399	♀	52	68	±	170	340	No viosterol 2 wks.	+	170	400	Rapidly worse
396	♀	52	53	++	110	330	No viosterol 2 wks.	+	150	500+	Rapidly worse, died
395	♀	41	78	+	160	320	No viosterol 1 wk.	+	180	480	Rapidly worse, died
394	♀	54	36	o	170	360	No viosterol 2 wks.	+	180	440	Rapidly worse, died
397	♂	49	125	o	60	160	No viosterol 2 wks.	+	210	320	Rapidly worse, died
331	♀	34		+	140	260	No viosterol 1 wk.	+	170	300	Rapidly worse, died
548	♂	35		++	200	500+	No viosterol				Moribund, died
547	♀	53		+	80	300	No viosterol				Moribund, died
606	♀	30		++	110	400	No viosterol				Moribund, died
25	♂	64	78	+	80	290	No viosterol				Moribund, died
681	♂	48		+	40	280	No viosterol				Moribund, died
708	♂	35	97	+++	200	500	No viosterol				Moribund, died
709	♀	45		+++	210	430	No viosterol				Moribund, died
804	♂	68	++	+	600+		No viosterol				Moribund, died
710	♀	53		o	80	300	No viosterol				Moribund, died

27 women. Forty-five of these patients showed no bleeding tendency. Thirty-eight of them were secondary carcinomata of the liver, 1 was a carcinoma of the gall bladder, 3 were carcinoma of the head of the pancreas, and 3 were carcinomata of the common bile duct. No viosterol was given. All were under observations for 2 to 7 weeks. All were either stationary or showed only a slow decline except 2 patients, 404 and 794. Patient 404 was a man 69 years old with an icteric index of 145. He had a low bleeding time but died apparently of cardiac decompensation. Patient 794 died although the bleeding time was normal.

Twenty-three of the 68 patients showed a bleeding tendency (Table VIII). There were 15 secondary carcinomata of the liver, 4 of the common bile duct, 3 of the pancreas, and 1 of the gall bladder. The Duke bleeding time averaged 150 seconds; the Ivy, 355. Eight of these were given viosterol. Three died, but 1 was moribund when treatment was started. Two became steadily worse, 2 remained unchanged, and 1 improved slightly. In this group, the Duke bleeding time remained at 130, the Ivy at 330.

TABLE VIII.—MEDICAL MALIGNANCIES WITH BLEEDING TENDENCY

Case number	Sex and color	Age	Diagnosis	Icteric index	Bleeding	Duke	Ivy	Viosterol	After viosterol			Result
									Bleeding	Duke	Ivy	
262	♀	54	Carcinoma of stomach	32	±	110	280	30 drops t.i.d. 2 wks.	o	80	160	Slightly improved; refused operation
721	♂	54	Secondary carcinoma of liver	30	o	30	250	30 drops t.i.d. 2 wks.	+		300	Steadily worse
348	♂	70	Secondary carcinoma of liver	130	+	130	290	60 drops t.i.d. 1 wk.	+	170	380	
								60 drops t.i.d. 1 wk.	+	200	420	
								5 drops 10,000X t.i.d. 2 wks.	±	50	300	
								5 drops 10,000X t.i.d. 2 wks.	o	80	280	Steadily worse
106	♂	89	Secondary carcinoma of liver	150	+	160	370	60 drops t.i.d. 1 wk. + calcium lactate	+	130	300	Died
105	♂	70	Secondary carcinoma of liver		o	100	280	60 drops t.i.d. 1 wk.	+	200	420	Died
476	♂	76	Secondary carcinoma of liver		3+	970		10 drops 10,000X t.i.d.				Moribund, died
799	♂	51	Carcinoma extra-hepatic bile ducts	4+	+	70	720+	30 drops t.i.d. + bile salts				Died
798	♂	61	Carcinoma extra-hepatic bile ducts	100	o	70	410	30 drops t.i.d. + bile salts 1 wk.		60	300	No change
722	♀	58	Carcinoma of gall bladder	90	++	80	430	No viosterol	+	100	410	Worse
410	♂	42	Secondary carcinoma of liver	110	o	50	140	No viosterol 1 wk. No viosterol 2 wks.	o +	170 150	240 330	Died
412	♀	52	Secondary carcinoma of liver		o	110	280	No viosterol 1 wk. No viosterol 2 wks.	o o	130 100	280 350	Died
413	♀	38	Secondary carcinoma of liver	86	o	90	260	No viosterol 1 wk. No viosterol 3 wks.	o +	100 100	340 380	Died
411	♂	65	Secondary carcinoma of liver		o	50	80	No viosterol	+		420	Died
403	♀	43	Carcinoma of gall bladder	52	o	70	200	No viosterol	o	100	260	Died
347	♀	38	Carcinoma head of pancreas	72	+	100	260	No viosterol	o	120	290	Died
720	♂	58	Secondary carcinoma of liver	50	4+	350		No viosterol				Died
475	♂	72	Secondary carcinoma of liver		o	120	600	No viosterol				Died
689	♂	59	Secondary carcinoma of liver	92	4+	90	390	No viosterol				Died
690	♀	52	Secondary carcinoma of liver	36	3+	210	420	No viosterol				Died
261	♀	60	Carcinoma of gall bladder	145	+	150	380	No viosterol				Died
797	♂	50	Carcinoma head of pancreas	73	o	120	310	No viosterol			740+	Died
796	♂	47	Carcinoma of common duct	4+	+	150	430	No viosterol		280	540	Died
795	♂	65	Secondary carcinoma of liver	+	+	80	560	No viosterol				Died
794	♂	70	Carcinoma of common duct	4+	+	50	50	No viosterol				Died

Fifteen patients were not given viosterol. Twelve died, but 5 were moribund on admission, leaving 6 deaths to compare with 2 of the other series. One patient became slightly worse; none showed even slight improvement. The Duke bleeding time remained at 130, the Ivy at 350. Viosterol was of no demonstrable value in these cases.

7. Cardiac decompensation with jaundice. There were 9 patients, 5 men and 4 women. Seven of them showed no bleeding tendency. All gradually improved under the usual cardiac therapy. One case was given viosterol but made no better improvement than the others. Two patients had prolonged bleeding time. One patient, 266, a man 41 years old, had an icteric index of 46, a Duke bleeding time of 150, and an Ivy bleeding time of over 600 seconds. He was moribund on admission. Viosterol was not given. He died the next day. One patient, 414, a man 57 years old, with an icteric index of 36 and an Ivy

TABLE IX.—TOXIC HEPATITIS WITH BLEEDING TENDENCY

Case number	Sex and color	Age	Diagnosis	Icteric index	Bleeding	Duke	Ivy	Viosterol	After viosterol			Result
									Bleeding	Duke	Ivy	
107	♀	58	Luetic hepatitis	65	o	40	130	No viosterol 1 wk.	o	70	280	Getting worse
								No viosterol 2 wks	±	100	360	Getting worse
								30 drops t.i.d. 1 wk.	o	70	250	Improving
								60 drops t.i.d. 2 wks.	o	50	130	Recovered
110	♀	33	Toxic hepatitis	28	+	100	350	60 drops t.i.d. 1 wk.	o	80	150	Recovered
625	♂	18	Toxic hepatitis	100	o	60	350	30 drops t.i.d. 10 days	o		70	Recovered
726	♂	27	Toxic hepatitis	60	o	120	260	5 drops 10,000X t.i.d. 1 wk.	o		60	Recovered
550	♂	25	Toxic hepatitis		+	100	300	No viosterol	o	70	150	Recovered
57	♀	34	Toxic hepatitis	32	o	100	260	No viosterol	o	70	120	Recovered
786	♂	39	Toxic hepatitis	++	+	190	610	No viosterol	+		500+	Worse
787	♂	47	Toxic hepatitis	60	o	120	260	5 drops 10,000X t.i.d. 1 wk.	o		60	Improved; recurrence
			(Second admission)	38	++	210	410	30 drops t.i.d. 1 wk.	o		140	Recovery

bleeding time of over 600, was given large doses of viosterol (60 drops three times a day). He died in 3 days. Viosterol was of no value in these cases.

8. Toxic hepatitis. Fourteen patients with toxic hepatitis were studied, 4 women and 10 men. Six showed no bleeding tendency and recovered promptly. Eight had prolonged bleeding times (Table IX). The Duke bleeding time averaged only 88 seconds, but the Ivy averaged 310. Five were given viosterol. Three were not. All recovered promptly. The Duke bleeding time decreased to 65 seconds, the Ivy dropped to 110. Of course, no effect from viosterol was demonstrated. However, 1 patient, 107, a colored woman 58 years old, had a low bleeding time on admission. Viosterol was not given. She began to grow worse. Viosterol was then given; she improved rapidly. Patient 787 had a prolonged bleeding time which dropped to normal under viosterol therapy. Treatment was stopped and he left the hospital. He returned 1 month later with recurrent hæmorrhages and prolonged bleeding time. Viosterol again brought his bleeding time down to normal and stopped his bleeding.

9. Miscellaneous. There were 15 patients with jaundice with miscellaneous conditions such as pneumonia, portal thrombophlebitis, ascending cholangitis, pyelonephritis, miliary tuberculosis, and Banti's disease (Table X). Viosterol was given in most of the cases in which the Ivy bleeding time was prolonged. The results seemed to depend largely on the basic condition rather than on the jaundice. At any rate, there were too few similar cases in this series to warrant conclusions.

10. Pernicious anæmia. Seventeen patients with pernicious anæmia were studied, 7 women and 10 men. Eleven had no bleeding tendency and showed a prompt remission under liver therapy. Six patients showed a bleeding tendency (Table XI). The Duke bleeding time averaged 150 seconds; the Ivy 340. Four were given viosterol and 2 were not. All, however, showed a prompt remission. The Duke bleeding time fell to 60 seconds, the Ivy to 115.

11. Leucæmia. There were 11 patients with leucæmia. Three of these, chronic myeloses, had no bleeding tendency. Viosterol was not given. They improved slightly while under observation and left the hospital. Eight of the leucæmias showed a marked bleeding tendency (Table XII). The Duke bleeding time averaged 380 seconds; the Ivy 500 seconds. Six of the patients were given viosterol; 2 were not. The bleeding time went even higher in all and all patients died. Viosterol was of no value in these cases.

TABLE X.—MISCELLANEOUS MEDICAL JAUNDICED CASES WITH BLEEDING TENDENCY

Case number	Sex and color	Age	Diagnosis	Icteric index	Bleeding	Duke	Ivy	Viosterol	After viosterol			Result
									Bleeding	Duke	Ivy	
52	♀	40	Portal thrombophlebitis	18	++	70	320	30 drops t.i.d. 10 days	0	60	80	Recovery
60	♂	33	Ascending pyelonephritis	+	+	70	260	30 drops t.i.d. 1 wk.	0	70	160	Recovery
479	♂	38	Banti's disease	93	+	270	590	No viosterol	+			Died
789	♀	40	Ascending cholangitis	31	0	140	320	No viosterol	+	180	360	Died
807	♀	20	Pneumonia	37	0	160	340	30 drops t.i.d. 9 days	0	50	90	Recovery
808	♀	33	Pneumonia	32	0	130	340	No viosterol	+	130	380	Died
601	♀	35	Empyema of gall bladder		+	140	380	30 drops t.i.d. 10 days + bile salts	0	70	160	Recovery
356	♂	44	Empyema of gall bladder	32	0	80	370	No viosterol				Died

TABLE XI.—PERNICIOUS ANÆMIA

Case number	Sex and color	Age	Bleeding	Duke	Ivy	Viosterol	After viosterol			Result
							Bleeding	Duke	Ivy	
278	♀	36	+	280	500+	60 drops t.i.d. 1 wk. 60 drops t.i.d. 1 wk. 60 drops t.i.d. 1 wk. + liver	0 0 0	260 120 90	450 220 130	Remission. Bogg's, 5 min. Howell, 12 min. Platelets 130,000
277	♂	33	+++		290	40 drops t.i.d. 1 wk. 40 drops t.i.d. 1 wk. + liver	0 0		160 140	Remission Howell, 9 min. Platelets 210,000
480	♀	46	+	240	350	30 drops t.i.d. 2 wks. + liver	0	60	90	Remission. Bogg's, 4 min.
559	♂	37	0	200	280	5 drops 10,000X t.i.d. 3 days + liver	0	60	120	Remission. Bogg's, 4½ min. Platelets 160,000
727	♂	58	0	30	260	No viosterol, liver only	0	50	100	Remission
728	♀	54	0	160	370	No viosterol, liver only	0	40	100	Remission

12. Secondary anæmia. Fourteen patients with secondary anæmia were studied. Six of them showed a prolonged bleeding time (Table XIII). The results of viosterol therapy, however, were inconclusive.

13. Purpura. There were 13 patients with purpura; 3 patients with toxic purpura, and 1 of septic purpura showed normal bleeding time. Viosterol was not given; all recovered promptly. Nine patients had prolonged bleeding times (Table XIV). Of these 1 case of septic purpura improved, while another similar case did not improve under viosterol therapy. Two patients with toxic purpura were given viosterol and recovered. Of the 6 patients with thrombocytopenic purpura, 2 were not given viosterol. They grew slowly worse. Two were given viosterol; 1 gradually improved, the other died. The fifth case, 119, came in bleeding very copiously. Whole blood injections controlled the hæmorrhages, and the patient slowly recovered after a 4 weeks' illness. Then he had a recurrence. This time viosterol was given without blood injections; he stopped bleeding in 3 days and remained well while under observations for over a month. The value of viosterol was not demonstrated in these cases. The one prompt remission was gratifying but lack of duplication in other cases renders conclusions unjustifiable.

14. Endocarditis. Four patients with endocarditis were studied. One case of rheumatic endocarditis with no bleeding tendency improved spontaneously. The three patients with malignant endocarditis showed a marked

TABLE XII.—LEUCÆMIAS

Case number	Sex and color	Age	Diagnosis	Bleeding	Duke	Ivy	Viosterol	After viosterol			Result
								Bleeding	Duke	Ivy	
7	♂	54	Monocytic leucæmia	3+	820+		30 drops t.i.d. 2 wks.	4+	1000+		Died; platelets 1,290,000
191	♀	45	Lymphatic leucæmia	2+	210	400	30 drops t.i.d.1wk.+X-ray therapy	2+	240	400	Died; Howell, 7 min.
359	♂	18	Acute leucæmia	4+	300+		5 drops 10,000X t.i.d. 1 wk.	4+	400+		Died
360	♀	16	Acute leucæmia	4+	300	600+	60 drops t.i.d. 1 wk. 5 drops 10,000X t.i.d. 2 wks. 5 drops 10,000X t.i.d. 3 wks.	4+ 4+ 4+	330 340 380	600+ 600+ 600+	Died
117	♂	4	Acute leucæmia	4+	500+		30 drops t.i.d. 1 wk.	4+	500+		Died
118	♀	46	Myelog. leucæmia	3+	460		30 drops t.i.d. 5 days	4+	500+		Died; platelets 420,000
282	♀	31	Myelog. leucæmia	3+	260	500+	No viosterol				Died; platelets 380,000
486	♀	46	Lymphatic leucæmia	+	200	400	No viosterol				Died

TABLE XIII.—SECONDARY ANÆMIAS

Case number	Sex and color	Age	Diagnosis	Bleeding	Duke	Ivy	Viosterol	After viosterol			Result
								Bleeding	Duke	Ivy	
115	♂	26	Secondary anæmia	3+	160	350	30 drops t.i.d. 4 days	0	60	110	Improved
116	♂	33	Secondary anæmia	+	100	280	30 drops t.i.d. 3 days	0	50	130	Improved
6	♂	17	Erythroblastic anæmia	3+	80	280	30 drops t.i.d. 1 wk. 30 drops t.i.d. 2 wks. 30 drops t.i.d. 3 wks.	+ 0 0	70 70 5	260 140 80	Platelets 600,000 R.B.C. 1,900,000; W.B.C. 12,700
62	♂	3	von Jaksch's anæmia	2+	480+		15 drops t.i.d. 2 wks.	+	500+		Subcut. liver therapy R.B.C. 2,300,000 W.B.C. 28,000; died
40	♂	62	Nephritic anæmia	3+	1000+		60 drops t.i.d. 3 days				Died
188	♂	64	Secondary anæmia; bleeding gastro-intestinal carcinoma	3+	90	360	30 drops t.i.d. 2 wks.	+		180	Gastric resection; died in shock

bleeding tendency. Two were given viosterol, one was not; all died.

15. Other miscellaneous patients. There were 11 miscellaneous patients without jaundice. These included patients with hæmophilia, Bantis' disease, scurvy, pellagra, tularæmia, ascites, etc. (Table XV). Viosterol was given to all patients with a bleeding tendency but there were too few similar cases to warrant conclusions. One patient with dermatographism had no bleeding tendency but was given viosterol to observe its effect. There apparently was no effect. The patient had a severe attack of "hives" while under treatment.

It is interesting to note that in 5 apparently normal individuals who were found to have a prolonged bleeding time (Table XV), the bleeding time returned to normal after from 2 to 5 days of viosterol administration.

SUMMARY AND CONCLUSIONS

There is no single method of predicting a bleeding tendency which is applicable to all types of hæmorrhagic diatheses. In the various types of bleeding dyscrasias, it is probable that various factors are responsible, and that different tests are required to demonstrate the bleeding tendency in each. Thus, in purpura the platelet count, in hæmophilia the Howell method of coagulation time determination, are the most reliable methods of predicting a bleeding tendency. In jaundice or in any liver deficiency, neither of these methods nor the Biffi, nor Bogg, nor Lee and White method of

TABLE XIV.—PURPURAS

Case number	Sex and color	Age	Diagnosis	Bleeding	Duke	Ivy	Viosterol	After viosterol: Bleeding	After viosterol: Duke	After viosterol: Ivy	Result
125	♀	17	Septic purpura	+	120	300	30 drops t.i.d. 2 wks.	o	30	120	Recovered
122	♂	14	Septic purpura	+	130	320	30 drops t.i.d. 2 wks.	+	170	350	No improvement
279	♂	40	Toxic purpura	+	100	280	30 drops t.i.d. 1 wk.	o	60	110	Recovered
11	♀	65	Toxic purpura	+	70	260	30 drops t.i.d. 1 wk.	o	40	70	Recovered
416	♀	24	Thrombocytopenic purpura	+++	80	290	No viosterol 1 wk. No viosterol 2 wks.	+++ 4+	90 140	260 290	Steadily worse; platelets 80,000
485	♂	36	Thrombocytopenic purpura	++	260	390	No viosterol 1 wk. No viosterol 2 wks.	+ 3+	250 280	370 380	Steadily worse; platelets 60,000
59	♂	24	Thrombocytopenic purpura	++	80	300	30 drops t.i.d. 2 wks.	o	80	120	Improved; platelets 150,000
119	♀	14	Thrombocytopenic purpura Bleeding returned platelets 95,000 No further bleeding for a month of observation	4+ 2+	600+ 120	1000+ 390	No viosterol; blood injections No viosterol; blood injections 3 wks. 30 drops t.i.d. 3 days	1+ o o	170 80 50	280 120 110	Boggs 3, Howell 7; platelets 40,000 Platelets 180,000 Platelets 190,000
120	♀	53	Thrombocytopenic purpura	4+	1000+		60 drops t.i.d. 1 wk.	4+	1000+		Died; platelets 40,000
764	♀	27	Thrombocytopenic purpura	4+	510	670	30 drops t.i.d. and 3 blood transfusions	o	90	180	Splenectomy; platelets rose from 70,000 to 150,000 before operation; recovery

TABLE XV.—MISCELLANEOUS

Case number	Sex and color	Age	Diagnosis	Bleeding	Bogg's	Duke	Ivy	Viosterol	After viosterol: Bleeding	After viosterol: Duke	After viosterol: Ivy	Result
189	♀	58	Hæmophilia	4+		180	290	30 drops t.i.d. 1 wk. + calcium lactate + hæmo-s 30 drops t.i.d. 1 wk. + calcium lactate + hæmo-s 30 drops t.i.d. 1 wk. + calcium lactate + hæmo-s 30 drops t.i.d. 1 wk. + calcium lactate + hæmo-s	3+ 1+ 2+ 3+	150 90 130 170	320 250 340 410	Steadily worse; Howell, 35 min.
9	♂	50	Dermatographia	o		80	50	30 drops t.i.d. 1 wk. 30 drops t.i.d. 2 wks. 30 drops t.i.d. 3 wks.	o o o	130 60 50	170 90 90	No improvement; had attack of hives under treatment
15	♂	29	Normal (threshold)	"Normal" o	4½	Subjects 240	370	30 drops t.i.d. 5 days	o	100	140	Satisfactory
16	♂	32	Normal (threshold)	+	5	80	240	30 drops t.i.d. 5 days	o	80	120	Satisfactory
17	♂	33	Normal (threshold)	o	4	50	230	30 drops t.i.d. 3 days	o	60	100	Satisfactory
18	♂	35	Normal (threshold)	o	6	70	260	30 drops t.i.d. 3 days	o	60	130	Satisfactory
19	♂	15	Normal (threshold)	+	5½	70	190	30 drops t.i.d. 2 days	o	50	100	Satisfactory

determining the coagulation time, nor the ordinary Duke method of determining the bleeding time, nor the fibrinogen content, nor the prothrombin content, nor the total calcium nor the sedimentation rate is reliable.

Among many modifications of the Duke test and other tests presumed to determine the bleeding tendency, the only one which we found to be reliable in cases of hepatic deficiency, was an application of the Duke method after

a pressure of 40 millimeters of mercury was applied to the arm. The technique of this test was standardized by trying it on 115 normal controls. This test, the Ivy bleeding time, does not normally exceed 240 seconds.

In the hæmorrhagic diathesis, the Ivy bleeding time practically always exceeds 240 seconds provided there is active or latent tendency to hæmorrhage. In the hepatic cases, especially in the face of active or latent tendency to hæmorrhage, the prolonged Ivy bleeding time is usually the only indication that trouble may be experienced. All the other accepted tests are usually negative.

The prolonged Ivy bleeding time and the hæmorrhagic diathesis of hepatic cases does not seem to depend so much on the intensity of the jaundice but rather on the degree of hepatic insufficiency. In this respect, the prognostic value of the Ivy test was striking.

When the Ivy bleeding time was within normal limits in hepatic cases, the prognosis was good. The medical cases almost always improved or recovered under observation even without specific treatment. The surgical cases almost always were operated upon without difficulty, and even without any particular pre-operative preparation made uneventful postoperative recoveries.

When the Ivy bleeding time in hepatic cases exceeded the normal limits, the prognosis was bad. If these cases were not treated, or if even with careful preparation and treatment the Ivy bleeding time remained prolonged, the medical cases usually became progressively worse and the surgical cases ran stormy postoperative courses or died. These results refer to hæmorrhage and hepatic insufficiency, and not to occasional cardiac, pulmonary, or other extrahepatic complications.

The prognostic reliability of the Ivy bleeding time is supported by the results observed in 810 hepatic and miscellaneous cases. The physiological mechanism underlying the test is probably the apparent fragility of the clot in hepatic insufficiencies. The coagulation time is frequently normal in these cases, but the clots yield to smaller pressure than do normal clots, and 40 to 60 millimeters of mercury pressure is sufficient to cause lack of fixation of the clot or to detect a latent failure of the small blood vessels to contract and retract normally. This would account for the fact that the Duke bleeding time is frequently normal in these cases while the Ivy modification of the test is prolonged. Gross experimental observation supports the hypothesis of clot fragility in hepatic deficiency. Further support is added by the clinical observation that these patients do not bleed abnormally at operation but after operation while the patient is thrashing around in post-anæsthetic recovery or is retching and vomiting, the hæmorrhagic catastrophes occur and transfusions become imperative. The effect of ether on the liver also may be here concerned and appear only after operation.

Viosterol 250D in doses of 30 drops three times a day supported by bile salts only in the presence of acholic stools almost always succeeded in reducing the prolonged Ivy bleeding time in cases of jaundice or hepatic deficiency to within normal limits in from 4 to 7 days, provided the hepatic deficiency was not too far advanced or fulminating in its progress. With the reduction in the Ivy bleeding time, the hæmorrhagic tendency would subside and the clinical condition improve.

The most plausible hypothesis in explanation is that in hepatic insufficiency there is difficulty in absorbing fat soluble vitamins, and the deficiency in the latter aggravates the bleeding tendency. When an excess of fat soluble vitamin is exhibited in the form of viosterol, this deficiency is corrected. In complete acholia, the absorption of viosterol is practically completely inhibited. The administration of bile salts then permits the absorption of viosterol.

Whatever the explanation, the clinical results obtained in the course of this study lead to the following conclusions:

1. Before cholecystectomy or choledochotomy if the Ivy bleeding time is normal, the prognosis is good even without specific preparation. If the Ivy bleeding time is prolonged, the operative prognosis is bad. In these patients viosterol for 4 days to 2 weeks will usually reduce the bleeding time to normal and insure a smooth postoperative recovery. Without viosterol, the bleeding time remains prolonged; the operative sequelæ grave.

2. Before exploration for probable malignancy in and about the liver the same results apply unless liver damage is too far advanced.

3. In medical cases of gall-bladder disease or common bile duct stone viosterol therapy is also indicated, if the Ivy bleeding time is prolonged. It then has some value, but its effect is not as striking as in the surgical cases.

4. In catarrhal jaundice, viosterol hastens recovery only in case of prolonged bleeding time.

5. In cirrhosis of the liver, viosterol is indicated in patients with a prolonged Ivy bleeding time and is of value provided the hepatic damage is not too far advanced or too rapid in its progress.

6. In the medical cases of malignancy in and about the liver, viosterol has no particular therapeutic value.

7. Viosterol was of no demonstrable value in the miscellaneous jaundices due to cardiac decompensation, pneumonia, toxic hepatitis, Banti's disease, portal thrombophlebitis, etc.

8. Viosterol was of no particular value in the bleeding tendencies resulting from the blood dyscrasias such as pernicious anæmia, leucæmia, secondary anæmia, hæmophilia, etc., nor was its value sufficiently demonstrated in our small series of purpura, scurvy, endocarditis, and miscellaneous bleeding cases.

9. Before tonsillectomy, bleeding histories may be checked by bleeding time determinations but our series of positive bleeders was too small to warrant conclusions.

10. The data on the relationship of the prolonged Ivy bleeding time and the effects of viosterol to blood chemistry changes will be reported in a separate communication.

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MECHANICS OF THE PHYSICAL SIGNS IN LOWER TRUNK INJURIES

LEOPOLD BRAHDY, M.D., NEW YORK, NEW YORK
From the Division of Workmens Compensation, Office of the Corporation Counsel of the City of New York

MOST injuries of the lower trunk involve the soft tissues only, are not demonstrable in roentgenograms, and must be studied clinically (10). The diagnosis of the lesions in which roentgenograms are negative is important from a therapeutic standpoint. The various results of trauma call for different treatment and thereby necessitate differential diagnosis. In compensation cases accurate diagnosis is important in order to distinguish between the malingerer and the honest claimant.

This paper is limited to the lower trunk injuries which involve the ischiogluteal bursa, the sciatic nerve, the ligaments and joints of the lumbar spine, the sacrum, the coccyx, and the hip. In all these injuries there is pain on motion of the trunk and of the extremities. To ascertain what the condition is, to differentiate it from hysteria and from malingering, we must analyze the pain producing and the pain relieving motions. A specific pain producing motion is usually named after the man who originally described it as a sign of one pathological condition. Thus flexion of the straight leg at the hip joint elicits pain in the dorsum of the thigh in sciatic neuritis, and is called Lasègue's (9) sign. The same motion elicits pain in the sacro-iliac region in sprain of the sacro-iliac ligaments and is then called Goldthwaite's (8) sign. Outward rotation of the semiflexed thigh may be limited and painful in lesions of the hip joint and is then called Patrick's or Laguère's sign. But the Patrick-Laguère motion may produce pain in sacro-iliac and in lumbar sprain. We have learned that, with few exceptions, the signs are not pathognomonic. Errors in diagnosis or failure to gauge the extent of the disability frequently are due to a misunderstanding of the physiology of the body motions used in the examination. The purpose of this article is to describe the physiological basis of the examination. A brief review of the pathology is first presented.

Sprain of ligaments. The function of a ligament is to *limit* motion of a joint. It follows that the ligament must be just long enough to permit the physiological motion, and to endure a certain amount of stress when there is any force attempting to exceed the physiological motion of the joint. In contradistinction to muscle tissue, ligaments have a minimal power of elongation. When a force is sufficient to overcome the resistance of the ligaments, there is an injury to the ligamentous tissue which we call a sprain. The longer the ligament the greater is the degree of motion beyond the physiological required to produce sprain. Where the motion between two bones is very small and the connecting ligaments short, the amount of movement beyond the normal required to strain that ligament is very slight. In a ligament like the lumbosacral, where the amount of physiological motion is small, a sprain can be produced by a motion forcing the ligament to stretch as little as an eighth of an inch. If the ligament is long, as in the ankle joint, it permits a wide range of motion, and several degrees of motion in excess of the normal are required to injure the ligament. The severity of sprains varies. Some give mild pain for a few minutes only; others give pain for months.

Pathological examination in the early stages of sprain shows tearing of some of the fibers of the ligament, with bleeding; subsequent examinations show the usual stages of inflammation and repair with scar tissue formation. If there is much scar tissue, its contraction results in shortening of the ligament. The shortened ligament will decrease the range of motion which was possible before the injury.

The bases for diagnosis of sprain of a ligament anywhere in the body are: (1) an area of swelling over the ligament; (2) a definitely localized point of tenderness over the ligament; (3) a spasm of the muscles which prevents stress on that ligament; (4) relief of pain when assuming a position which relieves that ligament of stress; (5) production of pain on

assuming a position which puts stress on that ligament.

In back sprains the swelling is sometimes present in the early stages. More often it is absent, because the ligaments are located beneath the muscle and fat tissue, so that the swelling is not perceptible. For the same reason, the tenderness is not as sharply localized as in the case of sprains of the more superficial ligaments of the extremities. The third, fourth, and fifth criteria of ligament sprain are the same in the back as elsewhere, but assume added importance because swelling and tenderness are unreliable signs.

The diagnosis of sprain is incomplete unless it includes a localization of the involved ligament. Sprains of three sets of ligaments of the lower spine produce signs which clearly differentiate them from one another: (1) the coccygeal; (2) the sacro-iliac and iliolumbar; (3) the lumbar.

Schmorl has established the importance of the intervertebral disks in the pathology of the spine. If the lesion in some traumatic spine injuries is in the intervertebral disks and not in the connecting ligament, the important physical signs must be similar. Functionally the disk is an elastic ligament connecting two vertebral bodies. As in sprain of the other intervertebral ligaments, pain is elicited when these two bodies move on one another. The motions causing or relieving pain will be similar to those of sprain of the true ligaments between the two vertebræ.

Ghormley has recently described injury to the cartilage of the articular joints of the spine, a true traumatic arthritis. He believes that the sudden onset of symptoms with "locking" indicates injury to the cartilage. The occasional sudden relief of symptoms on manipulation is also indicative of an intra-articular lesion rather than a ligament sprain. Most authorities believe that traumatic arthritis is an exceptional complication.

The controversy as to whether there is any such thing as traumatic subluxation of the sacro-iliac joint has continued for decades, sometimes merrily and often acrimoniously. As there is no direct evidence for its existence, I prefer not to use the term subluxation. However, there is no doubt that there is a pathological condition following sprain, the signs and symptoms of which are referable to the area of the sacro-iliac joint. In such cases it is found that any motion or position which puts stress on the ligaments of the sacro-iliac joint elicits pain. Any force that puts stress on the ligaments can also produce that minute motion which we know normally exists in this joint. Can the pain be due to sacro-iliac arthritis? Possibly—but motion so minute as to be demonstrable only in the dissected specimen does not elicit pain in any other joint except in fulminating inflammations. That the lesion is localized in the ligaments is the most reasonable hypothesis. Opinion on the pathology may differ. The signs and symptoms constitute a clear cut clinical entity.

It must be remembered that nearly every motion that puts stress on the sacro-iliac ligaments also puts some stress on the iliolumbar ligament. The signs and symptoms, which we call the sacro-iliac syndrome, may be due to strain of the iliolumbar ligament alone or in combination with strain of the sacro-iliac ligaments. The name sacro-iliolumbar sprain is more accurate. Nevertheless the name sacro-iliac syndrome for a combination of physical signs has been in use so long that a change in the name is not desirable.

Synovitis and peri-arthritis. The synovial membrane is a thin sheet of endothelium-like cells which invests the inner side of the capsular ligament of all true joints. Direct trauma (a blow) or indirect trauma (excessive stress on the capsular ligament) may produce an aseptic inflammatory process in this membrane. Synovitis frequently is manifested by an exudate in the joint. Usually the exudate is absorbed completely in time, and the joint resumes its normal function. In more severe cases, the exudate is fibrinous and the capsular ligament adheres to the joint surfaces resulting in painful and decreased range of motion. As the synovial membrane is intimately connected with the inner side of the capsular ligament, it is not possible to have a traumatic synovitis without some injury to the ligament. The ligamentous injury may resolve before the synovial exudate is absorbed so that clinically only the synovitis is of importance. On the other hand, the injury to the capsular ligament

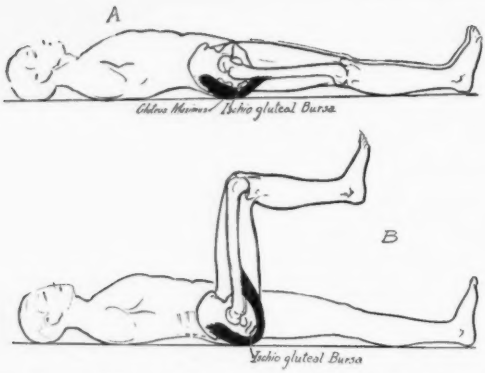

Fig. 1. Effect of flexion of the thigh on the gluteus maximus muscle; the relation of the muscle to the ischio-gluteal bursa is shown.

and the neighboring ligaments may give serious symptoms so that the amount of joint fluid and its absorption are secondary. The inflammatory reaction to trauma in the joint ligaments constitutes a peri-arthritis. In some joints the presence of fluid is easily demonstrated. In the interarticular joints of the vertebræ, in the sacro-iliac, and the sacrococcygeal joints such demonstration is not possible. In an occasional case of sprain of the hip ligaments, a fullness in the groin suggests fluid in the hip joint. In general, the only clinical evidence of synovitis in lower trunk injuries is pain when the involved joint is moved. But pain results from the same motion if there is traumatic inflammation of the capsular ligament of that joint. Clinically, no distinction can be made between synovitis and peri-arthritis *in these joints*. The clinical diagnosis of peri-arthritis, therefore, includes the signs and symptoms of any associated synovitis.

The sacrum and coccyx are separated by a fibro-elastic disk analogous to the intervertebral disk of the movable vertebræ. A synovia-lined joint is sometimes present and at times absent. Traumatic coccygodynia is a sprain of the sacrococcygeal ligaments. Had we any means of knowing that there is a true joint space in a particular case, it would be called a peri-arthritis.

Post-traumatic neuritis. The pathology and the mechanism of the production of traumatic neuritis are not definitely known. In 36 of 47 cases examined, the history or physical examination indicated that it was a sequel of lumbar or sacro-iliac sprain. This is approximately the proportion reported by others (4). Some authors (13) regard it as a radiculitis, and I believe this is correct. I use the term sciatic neuritis in the sense of an organic affection of the sciatic nerve.

Bursitis. Ischiogluteal bursitis is an inflammation of the lining of the bursa lying on the tuberosity of the ischium under the gluteus maximus muscle. The lining membrane is similar to the synovial membrane of a joint. There is usually fluid in the injured bursa.

EXAMINATION

It is unnecessary to discuss the importance of a general examination to exclude non-traumatic lesions. Many visceral diseases give rise to referred pain in the back. The outstanding characteristic of referred pain is that it is not dependent upon motion or posture of the body (16) in sharp contrast with the pain of lower trunk injuries.

The contours of the body, the gait, the manner of sitting down and of getting on the examining table must be observed. A spastic muscle by its pull will determine certain anatomical deformities, such as scoliosis in unilateral lumbar muscle spasm. Besides the deformities there are functional changes in posture. The patient will avoid pain by holding his body in such a position as to put the least tension or pressure on the injured tissue. Posture may be defined as the unconsciously assumed position of relief. The postures assumed in some of the lesions under discussion are quite characteristic and should be studied in conjunction with the positions of relief and of aggravation found on examination.

The active and passive motions of the trunk and extremities are tested. The important data to ascertain are: which motions elicit and which relieve pain; at what angle of motion the first pain is elicited; and what degree of limitation of motion is present. In examining sprains of some joints, such as the ankle, it is a simple matter to find the motions which elicit pain and those which relieve it. In examining for lower trunk injuries the motions producing relief and aggravation of the pain are more

complicated, as their effects are indirect. In the following descriptions the motions are grouped to aid in understanding their direct and indirect mechanical effects; the sequence is not the one commonly used in a physical examination. In all unilateral lesions a description of a lesion on the left side is given.

COCCYGODYNIA

Inspection and palpation. In sitting, the patient at times puts his weight on one ischial tuberosity, and at times on the other. When he lies on his back on the examining table there is no tendency to flex the knee or hip.

External pressure over the coccyx produces pain. The pain is usually more marked the nearer the tip of the coccyx the pressure is applied because this means greater leverage for movement at the sacrococcygeal joint. On rectal examination some pain is elicited on pressure over the anterior surface of the coccyx. The sacrotuberous ligament may be palpated on each side as it passes laterally from the sacrococcygeal junction to the ischial tuberosity. Pressure on one or both of these ligaments may elicit pain because their lower fibers are continuous with the sacrococcygeal ligaments.

Determination of pain producing motions. Abduction, rotation, or hyperextension of the thighs or hyperextension of the spine have no effect on the coccyx and produce no pain.

With the patient on his back, the examiner gently flexes one thigh on the abdomen. This motion produces pain accurately referred to the coccyx. Flexion of the other thigh elicits the same pain. How does motion at the hip joint affect the region of the coccyx? The gluteus maximus muscle arises from the ilium, sacrum, and coccyx and is inserted in the upper part of the shaft of the femur. Thigh flexion stretches this muscle (Fig. 1) and pulls on the coccyx. This results in pain analogous to that produced by direct pressure over the coccyx. In the standing position flexion of the trunk produces some pain. Flexion of the trunk on the thighs has the same effect on the gluteus maximus as flexion of the thighs on the abdomen and, therefore, elicits the same pain. Resumption of the erect posture is even more painful. In resuming the erect posture the gluteus maximus muscle is not only stretched but there is an active contraction of the muscle resulting in increased pull on the coccyx. With the patient sitting, trunk flexion has a similar effect on the gluteus maximus muscle but the contraction of the gluteus maximus is not as strong and the pain, though present, is less severe.

The reason for the posture is obvious. The patient sits on one tuberosity to avoid direct pressure on the coccyx. He does not flex the thighs because this results in pull on the coccyx.

ISCHIOGLUTEAL BURSITIS (LEFT)

Inspection and palpation. The patient sits with the weight on the right ischial tuberosity, never shifting to the left. There may be some tendency to keep the left thigh less flexed than is normal in the sitting position. When the patient lies on the table, the legs are kept straight without any tendency toward flexion of the hip or knee.

With the patient lying face down, there is an area of tenderness over the left ischial tuberosity—none on the right. The inflamed ischiogluteal bursa usually contains sufficient fluid so that it is palpable on gentle pressure over the buttock. There is no tenderness over the region of the sacrococcyx, and rectal examination is negative.

Determination of pain producing motions. With the patient lying on his back on the examining table, flexion of the left thigh elicits pain which is referred to the buttock, but not definitely localized. Flexion of the opposite thigh produces no pain. Rotation or hyperextension of either thigh has no effect.

The ischiogluteal bursa is located between the gluteus maximus muscle and the ischial tuberosity. Flexion of the thigh stretches the gluteus maximus muscle and therefore puts pressure on the tender ischiogluteal bursa (Fig. 1). Rotation and extension of the thigh put no pressure on the bursa, and therefore have no effect.

In the standing and in the sitting position, flexion of the trunk produces the same pain as hip flexion because we have the same stretching of the gluteus maximus muscle with the resulting pressure on the ischiogluteal bursa.

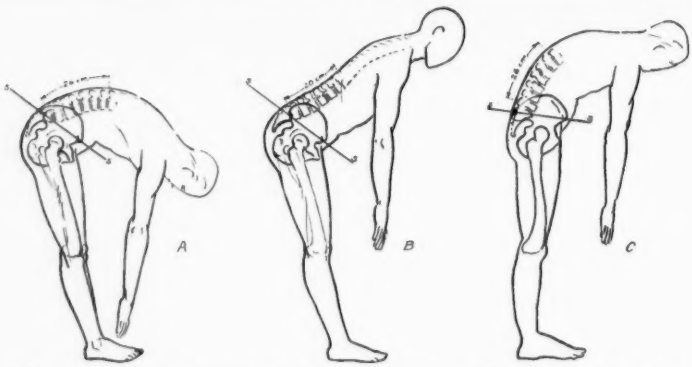

Fig. 2. Flexion of the trunk in standing postion. A, Normal trunk flexion by movement of both spine and pelvis. B, Partial flexion of the trunk by pelvic rotation but without movement of the spine. C, Partial flexion of the trunk by spinal motion but without pelvic rotation. The angle of line *S-S* shows the degree of flexion of the pelvis.

The reason for putting the weight on the unaffected side, in sitting, is because of the tenderness of the bursa located directly on the ischial tuberosity. The patient does not flex the left thigh completely because complete flexion stretches the gluteus maximus muscle.

MECHANICS OF NORMAL MOTIONS OF THE TRUNK

Trunk flexion is composed of two simultaneous movements: flexion of the spine and flexion of the pelvis (Fig. 2). In flexion of the spine the normal lumbar lordosis disappears, and changes to a gradual, smooth, convex curve (Fig. 2A). In flexion there is very little movement of the thoracic spine, practically all of the motion taking place in the lumbar portion of the spine. The amount of motion in the lumbar spine can be determined more accurately with a tape as described by McKendrick. A horizontal line is drawn across the eleventh thoracic vertebra, and a parallel line across the first sacral vertebra. With full lumbar spine flexion the distance between these two lines is increased more than 6 centimeters.

Flexion of the pelvis takes place at the hip joint. As part of the pelvis is below the pivot of motion this movement may be more accurately described as a forward rotation of the pelvis; the axis of rotation is a line connecting the two acetabula. In backward movement of the trunk (extension), the pelvis moves in the opposite direction and the motion is called backward rotation of the pelvis.

Flexion of the spine and forward rotation of the pelvis contribute about equally to complete trunk flexion. The pelvis can be rotated forward without any motion of the spine (Fig. 2B) resulting in a partial flexion of the trunk. The absence of spinal movement may be voluntary, it may be due to pain on spinal movement or it may be the result of ankylosis of the spine. Partial trunk flexion may be effected by flexion of the spine alone (Fig. 2C). The absence of forward rotation of the pelvis may be due to ankylosis of one or both hips, or to pain on forward rotation of the pelvis as in peri-arthritis of the hip joint. We have seen that forward rotation of the pelvis, by stretching the gluteus maximus muscle, produces some pain in ischiogluteal bursitis and in coccygodynia. There may be a tendency to limit pelvic rotation in these two conditions, but there will be no limitation in flexion of the spine.

In every active motion there are two factors—the contraction of one muscle (or group) and the associated relaxation of its antagonist. The normal range of motion, whether passive or active, is usually limited by the structure of the joint surfaces and by the length of the joint ligaments. Some motions are limited by the length of the muscle antagonist. With the patient on his back, passive flexion of the

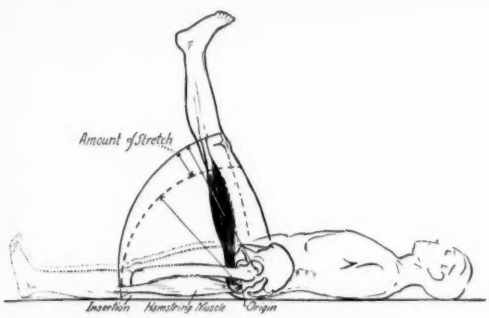

Fig. 3. Showing the stretching of the hamstring muscles by flexion of one leg. The dotted arc is the arc which would be described by the hamstring muscles if they were not forced to stretch. The solid arc is that described by the points of bony attachment of the muscles. The distance between the two arcs at any point is the amount the muscle must be stretched when the leg is flexed to that point.

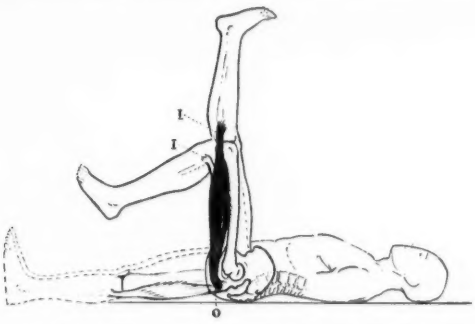

Fig. 4. Showing the decrease in distance from the origin, *O*, of the hamstring muscles to insertion, *I*, on flexion of the knee joint with resulting relaxation of the hamstring muscles.

straight leg on the abdomen is limited to 90 degrees by the normal hamstring muscles. As the examiner flexes the patient's straight leg the distance between the attachment and insertion of the hamstring muscle must increase (Fig. 3). To visualize this fact we must remember that in flexion the leg revolves with the head of the femur as a pivotal center. The hamstring muscles revolve with their point of origin on the tuberosity of the ischium as a center. Imagine the hamstring muscles detached from their insertion on the upper part of the tibia and fibula. If the leg and the detached muscle are revolved together the free ends of the muscles, because their pivotal center is posterior to that of the femur, describe an arc proximal to the arc described by the heads of the tibia and fibula as shown in Figure 3. With increasing flexion, there must be an increasing stretching of the muscle, if it is to extend to its point of attachment. When the straight leg has been flexed 90 degrees, the limit of elasticity has been reached. Any attempt to flex the straight leg beyond the limit of elasticity of the hamstring muscles produces muscle pain referred to the dorsum of the thigh. This movement tests the elasticity of the hamstring muscle. With the straight leg flexed, as shown in Figure 3, the hamstring muscle can be relaxed by bending the leg at the knee joint (Fig. 4) because this brings the origin and the insertion of the muscle closer together. The thigh can now be flexed completely on the abdomen.

Just as the hamstring muscles limit elevation of the leg, when the patient is lying down, so they limit forward rotation of the pelvis in the standing position. Flexion of the knee joint relaxes the hamstring muscles and makes greater pelvic rotation possible. If, however, pelvic rotation is limited by disease of the hip joint, the relaxation of the hamstrings will have no effect.

With one leg in the same position as in Figure 3, the hamstrings could also be relaxed by forward motion of the point of origin of the muscles on the ischium. This means a backward rotation of the pelvis. But with the right leg straight on the examining table, the iliofemoral ligament of the right hip is taut and permits only slight backward rotation of the pelvis. When the examiner flexes both legs at the hip, as in Figure 5, the hamstrings can pull the ischium forward. Therefore, a greater degree of flexion of the legs on the abdomen is attainable when the legs are flexed simultaneously than when one leg is flexed alone. The amount of movement at the hip joints is the same in the 2 cases but with simultaneous leg flexion the whole pelvis rotates backward. In this case the pull of a muscle causes motion in a part not directly moved by the examiner. In motions other than straight leg raising such indirect movement is seldom due to muscle pull but to ligament pull. When a muscle is not spastic or

shortened by disease, it stretches sufficiently to permit as much motion as the length of the ligaments will permit.

The sacrum is part of the pelvis. Backward rotation of the pelvis means a similar motion of the sacrum. This produces a movement of the lumbar spine. The normal concave lumbar curve is first flattened and then becomes convex (Fig. 5). This movement can be demonstrated by the examiner slipping one hand under the concavity of the lumbar spine as the patient lies on his back. When the two legs are flexed at the hip he will feel the pressure of the patient's back as the concavity disappears and the back is flattened. In a preceding paragraph it was shown that but little pelvic motion is possible if the examiner raises one leg alone. It follows that movement of the lumbar vertebræ occurs only when both legs are raised simultaneously. The longitudinal lumbar muscles do not take any part in this motion; they are passively stretched. Normally they are sufficiently elastic so that the vertebræ can move as far as the structure of the ligaments and bones will allow. The sacrum and the lumbar vertebræ are connected to one another by numerous ligaments. As soon as the pelvis begins to rotate there is a pull on the lumbosacral ligaments. As rotation increases the pull is transmitted through the connecting ligaments to the upper lumbar spine. This explains the finding that in lumbosacral sprains pain is elicited on moderate elevation of both legs and by increasing the amount of elevation, pain is elicited in sprains of the higher lumbar ligaments.

When we speak of motion of the pelvis it must be remembered that though it moves as one piece, the pelvis is not a solid bone. It is composed of three bones, the right and the left innominate bones being united to the sacrum by powerful ligaments. In the erect position most of the weight of the trunk falls on the sacrum and is transmitted through the sacro-iliac ligaments and the innominate bones to the lower extremities. If the sacro-iliac ligaments are strained, pain will be felt in the area of the joints in the normal standing position. Although there is some cartilaginous and bony connection between the sacrum and the ilium, the sacro-iliac articulation has a joint space and a minute amount of motion in the joint can be demonstrated in the cadaver. A force exerted through the pelvis (from the trunk to the extremities or from the extremities to the trunk) must be transmitted through the sacro-iliac ligaments. Any motion which tends to rotate the pelvis backward or forward must result in some stress on these ligaments.

In the standing position, on flexion of the trunk the pull of the hamstring muscles on the ischial tuberosities tends to pull the pelvis backward. The weight of the trunk exerts a forward stress on the upper sacrum, thus tending to rotate the pelvis forward. The resultant of these two opposing forces is a torsion stress on every part of the pelvic ring between the ischium and the sacrum. This ring is composed of bone except at the sacro-iliac joints and the symphysis, and, if the ligaments are sensitive to pull, marked pain will result.

With the patient lying prone on the table, flexion of the knee stretches the rectus femoris muscle. The origin of this muscle is on the anterior spine of the ilium. The muscle pull tends to rotate the pelvis forward. The stress of the motion is transmitted through the sacro-iliac ligaments to the sacrum and through the lumbosacral ligaments to the lower spine. This movement, therefore, elicits pain in sprain of either of these ligaments but puts no strain on the sciatic nerve, the ischiogluteal bursa, or the coccyx.

With the patient again on his back, the heel is placed on the opposite patella, and the thigh is rotated outward and abducted (Fig. 6). In arthritis of the hip joint, pain will be elicited because of the movement in the joint. This motion stretches the psoas magnus muscle which takes its origin from the bodies and the transverse processes of the five lumbar vertebræ and is inserted into the lesser trochanter of the femur. By muscle pull on the lumbar vertebræ, slight motion of the vertebræ is produced, putting some stress on the intervertebral ligaments. When abduction is complete, the hip ligaments are under tension and the pelvis is rotated around its vertical axis (circumduction). This rotary

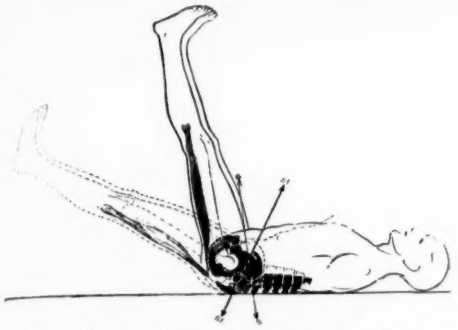

Fig. 5. The hamstring muscles rotate the pelvis backward to the position shown in solid color. This pelvic rotation flattens the lumbar curve (which is the equivalent of flexing of the lumbar spine). Backward rotation of the pelvis brings the origin of the hamstrings forward, thus relaxing the hamstring muscles and permitting greater flexion of the thighs on the abdomen. The arrows pass through the anterior superior spine and the posterior superior spine of the pelvis. *S-S*, First position; *S'-S'*, after flexion of legs with rotation of the pelvis.

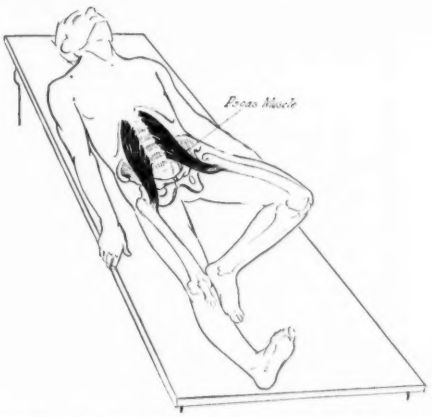

Fig. 6. The left heel is on the patella of the opposite leg. Outward rotation and abduction of the thigh is done by depressing the left knee. The resulting stretch of the psoas magnus muscle is shown.

stress is transmitted through the sacro-iliac ligaments to the sacrum and through the lumbosacral ligaments to the lumbar spine. If any of these ligaments are sensitive, pain will be elicited. This motion puts no stress on the sciatic nerve or on the coccyx.

Let us return to a consideration of stress on sacro-iliac ligaments. With the patient lying face down, the knee is raised 12 centimeters from the table. This is approximately the limit of hyperextension at the hip joint, and on attempting further motion the stress is transmitted through the hip ligaments to the pelvis. The sacro-iliac ligaments of the same side feel the stress first. It is then transmitted through the sacro-iliac ligament of the same side, via the sacrum, to the sacro-iliac ligament of the opposite side. Pain on this motion, therefore, is elicited on the opposite side, if the opposite sacro-iliac ligament is sensitive.

The patient then lies on the left side with the legs straight, and the examiner hyperextends the right thigh. Stress is felt in the opposite sacro-iliac ligament, but the stress is dissipated by motion of the pelvis with associated increase of the lumbar lordosis. This motion of the lumbar spine is, of course, transmitted through the lumbar ligaments. If the left leg, instead of being straight on the table, is completely flexed at the hip and knee, motion of the pelvis is prevented. The stress of the motion cannot be translated into the movement of the pelvis, but is concentrated on the sacro-iliac ligaments of both sides. In this position, therefore, sacro-iliac pain will be more acute and lumbosacral pain will be mild. When the leg is extended and pelvic motion is possible, sacro-iliac pain will be mild while lumbar ligament sprain will elicit marked pain.

LESION OF THE HIP JOINT

Inspection and palpation. The patient sits with his weight on the right ischial tuberosity. He sits near the forward part of the chair so that the left hip is slightly flexed but not at a right angle. The posture when lying on the examining table is quite characteristic—the left thigh slightly flexed and everted and the knee slightly bent. Occasionally a slight fullness is noticed below the middle of Poupart's ligament. There may be tenderness at this site. With the left leg straight, a blow on the heel may produce pain in the region of the hip joint; this is a valuable sign but seldom present in traumatic lesions.

Determination of pain producing motions. With the patient lying on his back, the straight leg of the affected side is rotated outward. This movement elicits pain, a sign absent in any of the other conditions with

which we are dealing. This movement stretches the anterior hip ligaments and the anterior portion of the hip capsule. There is not sufficient leverage for the force of this motion to be transmitted to any other joints or structures.

Although slight flexion and outward rotation is the position of relief assumed by the patient, there is pain when flexion with outward rotation is carried further. This is tested by putting the heel of the affected leg on the opposite patella and pressing the knee outward (Patrick-Laguère sign). This motion produces pain in peri-arthritis of the hip in the same manner as rotation of the straight leg, except that, the force being very much more marked, it is positive in cases where the stress of the outward rotation of the straight leg is not sufficient to produce pain. The force of this motion is sufficient to be transmitted to the whole of the pelvis and may produce pain in other conditions besides lesions of the hip joint.

Lesions of the hip joint are often associated with spasm of the psoas magnus muscle. The muscle is stretched by the movement just tested—outward rotation and abduction of the semiflexed thigh (Fig. 6). When this muscle is spastic the range of this movement will be decidedly decreased and painful. This is because any spastic muscle will not permit the same amount of stretching that the normal relaxed muscle permits. Stretching of any spastic muscle produces pain. Limitation of the range of outward rotation of the semiflexed thigh is, therefore, indicative of psoas spasm. Psoas spasm, however, is not diagnostic of lesions of the hip joint. It occurs also in sacro-iliac and lumbar sprains.

The patient now lies on the right (unaffected side) with the right leg straight. When the examiner hyperextends the left thigh, mild pain is produced. The right thigh and leg are now flexed against the abdomen and the left leg again hyperextended. More intense pain is now elicited. This difference in degree of reaction is due to the fact that when the right leg is straight the pull on the anterior ligaments of the left hip produces a forward rotation of the pelvis, thus changing some stress into motion. When the right thigh and knee are pressed against the abdomen, the pelvis cannot rotate forward, and the full force of hyperextension of the left thigh is felt in the ligaments of the left hip.

In the standing position there is some limitation and pain on flexion of the trunk. Observation and measurement of the spinal motion with a tape will show that this limitation is in the forward rotation of the pelvis and not in the flexion of the spinal column (Fig. 2C). In the sitting position the same limitation and pain on flexion of the trunk is noted.

The reason for the posture assumed is that hyperextension or marked flexion or marked eversion of the thigh each put stress on the hip ligaments and the position of relief is therefore a position of slight flexion and slight eversion. In sitting this can be done only by putting the weight on the opposite ischial tuberosity.

SPRAIN OF THE LUMBAR SPINE

Inspection and palpation. The normal lumbar lordosis is usually increased, especially in the acute stages with lumbar muscle spasm. In the chronic cases the curve may be flattened. There is no tilting of the spine but a mild lumbar scoliosis may be found with unilateral muscle spasm. The marked scoliosis found in sacro-iliac sprain and in sciatic neuritis is never present.

In the acute stages the muscles on one or both sides of the lumbar spine may be prominent, firm on touch, and the skin over the muscles may be very tender. Because of this diffuse tenderness, the pain elicited by pressure over the involved ligament may be masked. In the less acute stages the examiner may elicit tenderness to deep pressure at the sacrolumbar joint or between any two of the lumbar vertebræ. Often the tenderness is localized to one side of the midline indicating that the lesion is in one of the paired ligaments of the lumbar spine. The localization of the tenderness does not vary in the course of the examination. Variable tenderness suggests malingering or "neurotic" spine (2).

Determination of pain producing motions. In lesions of the lumbar spine, pain is elicited when the patient assumes any position which

produces motion of the lumbar vertebræ or at the lumbosacral joint because any such motion puts stress on the ligaments. The articular capsules and the ligaments of the bodies and of the arches form a complicated system. It may not be possible to discover which of these ligaments is involved in an individual case. The sprain is often in one of the paired ligaments so that the signs are more marked on one side. However, any motion which produces stress on the ligaments of one side will affect those of the opposite side to some extent. The pain on the two sides differs in intensity but not in the type of motion which produces it.

With the patient sitting on a stool, lateral bending of the trunk produces some pain in the lumbar region. Lateral bending is in large part a movement of the lumbar vertebræ, and the stress is felt in the ligaments connecting the vertebræ.

With the patient still sitting on a stool, the examiner grasps the shoulders of the patient and rotates the trunk. Rotation is a movement of the thoracic and cervical vertebræ, and little stress is felt in the lumbar vertebræ until partial rotation has taken place. This motion is therefore painful only in the second half of its range.

Both flexion and extension of the trunk are painful because this is, in part, a movement of the lumbar spine. Full flexion or extension may not be possible because of pain. In flexion of the spine the first movement is at the lumbosacral joint; with lesions of the ligaments connecting the fifth lumbar with the sacrum the pain is therefore felt on very slight motion. With increasing flexion of the spine the fourth, then the third, second, and first lumbar vertebræ take part. With lesions higher up, therefore, the pain is felt on increasing flexion. The higher the ligaments involved, the greater the amount of flexion before pain is produced.

Extension of the spine is examined with the patient prone on the table. The two thighs are simultaneously passively hyperextended. This produces a forward rotation of the pelvis, the stress falling first on the lumbosacral joint. As the thighs are raised more, the stress falls on the ligaments of the upper lumbar vertebræ. If hyperextension is done by raising the upper trunk instead of the thighs, the stress falls first on the uppermost lumbar ligaments and only with greater extension on the lumbosacral joint. Therefore, when slight extension of the thighs produces pain (lumbosacral sprain) marked extension of the upper trunk is necessary to produce the same reaction. In lesions of the upper lumbar vertebræ, marked extension of the thighs or else slight extension of the trunk are required to produce pain.

When the patient lies on his back and the two legs, with knees straight, are raised simultaneously, the motion during the first 20 degrees is a simple movement at the hip joint. With increasing flexion, the pelvis rotates backward (Fig. 5). The pelvis moves as one piece, and this backward rotation of the pelvis means that the sacrum is rotating in the same direction and pulling on the lumbosacral ligaments. If there is a strain of this ligament there will be pain on this motion. With increased backward rotation of the sacrum there is a flattening of the normal lumbar lordosis. The movement of the lumbar vertebræ is analogous to that which takes place in flexion of the lumbar spine. Flexion of the thighs, therefore, produces pain in sprain of any of the lumbar ligaments. Moderate flexion will produce pain in lumbosacral sprain. Increasing flexion produces pain in sprain of the ligaments of the higher lumbar vertebræ.

Flexion of the thighs produces pain in the lumbar region only in so far as this position produces backward rotation of the pelvis. There is only slight rotation of the pelvis on flexion of the thighs with the knees bent. When the legs are straight, more marked pelvic rotation occurs because of the pull of the hamstring muscles on the ischia. When the examiner's hand is placed beneath the upper part of the sacrum he can determine that the pain is never elicited before the thigh flexion causes some backward rotation of the sacrum. Pain due to rotation of the pelvis is the distinguishing feature of lumbar lesions.

The origin of the pain can be checked by another test. When the patient is lying on the left side with the left knee and hip held in flexion, hyperextension of the right thigh elic-

its but mild pain. With the left leg extended in the anatomical position, hyperextension of the right thigh produces much more pain. When the thigh and leg are flexed, the pelvis is fixed and the force is not easily transmitted to the lumbar spine, but when the left leg is extended the force is quickly translated into motion of the lumbar spine. The results in lumbar sprain are the reverse of those obtained in sacro-iliac sprain.

Outward rotation of the semiflexed thigh will produce pain if the motion is carried far enough because this motion causes a circumduction of the pelvis (rotation on its vertical axis) with its inevitably associated rotation of the lumbar spine. Pain on this motion may also be the result of pull of the psoas magnus muscle on the lumbar vertebræ (Fig. 6).

MUSCLE SPASM

In the acute stages of lumbar sprain there is spasm of the lumbar muscles. Usually bilateral, it may be unilateral. There may be no spasm with the patient at rest, but it appears only when the patient flexes the spine or (with the patient on his back) the examiner flexes the thighs.

The normal range of motion of the skeleton is partly dependent on the relaxation of an antagonistic set of muscles. Passive or active motion will be less than normal, if the antagonists are shortened by disease or by reflex contraction. This decrease in the range of motion, or limitation of motion as it is usually called, is important in the diagnosis of trunk injuries. A patient may limit a pain producing motion, even though no muscle rigidity is present. This voluntary limitation may be overcome by reassurance and gentleness on manipulation. Voluntary contraction is rarely limited to one specific group of muscles but is a general contraction of the muscles of the trunk or limb on motion. It may appear after the motion is completed. Involuntary muscle spasm is limited to specific muscle groups, is often (not always) found at rest as well as on motion, and is present or develops during the motion and not after the motion is completed. Voluntary contraction is not usually a sign of malingering if it occurs with a pain producing motion. If the same reaction occurs with a motion which should not be accompanied by pain we may conclude that the patient is malingering.

Motion which stretches a spastic muscle will be painful and the range of that motion will be limited. The determination of which motions produce pain and are limited because of muscle spasm, is essential for two reasons. First, the presence of spasm in specific muscle groups and its absence in other groups is indicative of certain underlying lesions. Second, these underlying lesions, themselves, are diagnosed largely on the basis of the tenderness and painful motions to which they give rise. The motion limited by a spastic muscle is always one which would cause pain in the underlying lesion if the motion were carried to its full range. The limitation of motion and the pain of muscle spasm are always evident before pain is produced at the site of the lesion itself. The pain of muscle spasm is referred to the site of the muscle and not to the site of the lesion.

Lumbar muscle spasm. The sacrospinalis muscle group is chiefly involved in this spasm but it seems probable that the multifidus, the semispinalis, and the small intervertebral muscles also take part. The sacrospinalis muscle is attached below to the sacrum, to the spinous and transverse processes of the lumbar vertebræ, to the lumbar fascia and the eleventh and twelfth ribs. It lies in a groove on each side of the vertebral column. It splits into various groups which are attached above to the lower nine ribs, to the spine, and to the transverse processes of the upper thoracic vertebræ. Some one fasciculus being attached to every bone of the trunk, there is hardly a motion of the trunk which does not stretch part of this extensive muscle group. In extreme spasm, deep breathing may be limited by the fibers attached to the ribs.

Palpation. The patient lies prone on the examining table. The hard, sharply outlined mass of a very spastic muscle is easily palpated but it is necessary to train the fingers to feel lesser degrees of spasm. Spastic muscle is always tender. The extent of the muscle involved, as well as degree of spasticity, varies with the severity of the lesion; in mild cases only a segment may be affected.

Determination of pain producing, limited motions due to muscle spasm. In lumbar spasm many motions may be limited; only the more important ones commonly used in diagnosis will be described. Flexion of the trunk is limited in so far as this is a movement of the spine, but that part of the movement which is due to forward pelvic rotation is not limited. The limitation of trunk flexion is entirely due to limitation of intervertebral movement. The limitation of lumbar spine movement is the same in sitting as in standing. It follows that the *difference* between flexion of the trunk in the standing, and in the sitting, posture is the same as in the normal individual. Not alone are movements between the lumbar vertebræ limited but also movements between the fifth lumbar and sacrum, because the sacrospinalis muscle is attached to the sacrum. Any backward rotation of the pelvis will stretch that muscle. This is the effect of flexion of the two thighs when the patient is lying flat on his back (Fig. 5). The backward rotation of the pelvis is more marked if there is forward pull on the ischia by the hamstring muscles, even though there is no hamstring spasm. We have discussed the fact that hamstring muscle pull is greater when the knees are straight. The pain and limitation of hip flexion will, therefore, be greater when the knee is straight and will be relieved when the knee is flexed. Because the pelvic rotation is very slight before the end of the first half of trunk flexion, the limitation is rarely as marked as in hamstring spasm. As the limitation is due to an effect on the lumbar muscles, the pain will be referred to the lumbar region instead of to the dorsum of the thigh. Hamstring tension is somewhat relieved on backward rotation of the pelvis, but this motion increases the tension on the lumbar muscles. It has previously been stated that raising the two legs simultaneously produces more pelvic rotation than raising one at a time. In lumbar spasm, therefore, raising both legs is more painful and limited than raising one leg at a time—in contrast to the result in hamstring spasm.

In unilateral spasm the pain will be present on the affected side only. Spasm of the hamstring muscles is occasionally found in lumbar lesions, but because it is much more common with sacro-iliac sprain and sciatic neuritis, it will be described later.

PSOAS MUSCLE SPASM

The psoas muscle may be spastic in lumbar and sacro-iliac ligament sprain as well as in hip conditions. The muscle lies too deep for palpation; spasticity must be determined entirely by indirect means. It is attached above, to the five lumbar vertebræ and is inserted below, into the lesser trochanter of the femur.

Determination of pain producing, limited motion. With the patient on his back, the heel of one foot is placed on the opposite patella. The thigh is then rotated outward and abducted (Fig. 6), thus stretching the psoas muscle. When the psoas is spastic this movement elicits pain referred to the pelvic region and the range of the motion is limited.

SACRO-ILIAC SPRAIN (LEFT)

Inspection and palpation. The spine is tilted forward and usually to the right, that is, toward the unaffected side. The normal lumbar lordosis is flattened. If scoliosis is present the convexity is to the left (homologous scoliosis). In standing, the weight is on the right leg; the left knee is slightly bent. There is no eversion of the leg at the hip. Standing is tiring and the patient prefers to sit, putting the weight on the right ischial tuberosity.

The left lumbar muscle may be spastic; occasionally there is spasticity of both lumbar muscles and rarely of the opposite side alone. There is tenderness to the left of the fifth lumbar vertebra and along the posterior border of the ilium, most marked near the posterior, inferior spine.

Determination of pain producing motions. The patient lies flat on his back. Flexion of the left thigh with the knee bent, produces a moderate rotation of the pelvis by pull on the hamstring and gluteal muscles. As the pull is on one half of the pelvis while the other half is fixed by the position of the right thigh, the movement produces a rotary stress on the sacro-iliac joint ligaments. When flexion is complete, there is some rotary movement of the pelvis on the lumbar spine. The ilio-

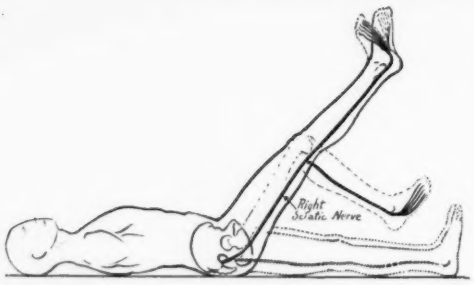

Fig. 7. Flexion of the straight leg results in a pull on the sciatic nerve trunk. Dorsiflexion of the ankle has the same effect. Flexion at the knee relieves the pull.

lumbar ligament is put under stress by this motion. Thigh flexion produces some pain in all sacro-iliolumbar conditions.

When the thigh is flexed with the knee straight, the hamstring muscles come into action at a much lesser angle of thigh flexion. The straight leg is flexed to the point at which moderate pain is produced. In this position bending the knee at right angles results in sudden cessation of pull on the pelvis by the hamstring muscles, and further flexion without pain is possible.

The patient now turns to lie face downward. Hyperextension of the left thigh elicits pain in the left sacro-iliac area. Hyperextension of the right thigh produces no pain at first and on more marked extension pain is produced on the opposite side. Contralateral pain on hyperextension is characteristic of sacro-iliac disease; it was first described by Gaenslen.

With the patient again on his back, the heel of the left foot is placed on the right patella (Fig. 6) and the left knee joint pressed downward and outward (i.e., outward rotation of the semiflexed thigh). When this motion is complete there is pressure by the femur on the left half of the pelvis with a shearing stress on the sacro-iliac joint. This motion, therefore, elicits pain in sacro-iliac ligament sprain. Similar action on the right side will elicit no pain or mild pain referred to *left* sacro-iliac area.

With the patient lying on the right side, the left thigh is hyperextended. This produces stress on the sacro-iliac ligaments, but before it can become very great the pelvis rotates forward. If the pelvis is fixed by complete flexion on the right hip and knee, the same hyperextension produces much more marked stress on the sacro-iliac ligaments. With the patient lying on the left side these maneuvers are repeated. When strain is put on the right sacro-iliac joint, there is no pain but when the pelvis is fixed, sufficient stress is transmitted to the opposite sacro-iliac area to produce pain.

In the sitting position, flexion of the trunk is painful because the pull of the flexed spine is transmitted through the sacrum to the sprained sacro-iliac ligament. Because of this pain the patient may limit the flexion of the lumbar spine but that part of trunk flexion due to pelvic rotation is not limited.

In the standing position, trunk flexion is more painful than in sitting. In both positions we have the weight of the flexed spine tending to rotate the sacrum forward. In standing, with legs straight, we have a second factor—the hamstring muscle pull preventing the full rotation of the pelvis forward. These two forces, one acting on each side of the sacro-iliac joint but in opposite directions, produce a marked stress on the sacro-iliac ligament. The pain and limitation of trunk flexion is, therefore, more marked in standing than in sitting.

Sacro-iliac sprain may be associated with spasm of one or more of three groups of muscles: the lumbar, the psoas magnus, and the hamstring muscles.

HAMSTRING SPASM

Inspection and palpation. Spasm of the hamstring muscles cannot be palpated at rest —it is always latent. Examination of the patient as he rests on the table is negative.

Determination of pain producing, limited motions. The patient lies on his back on the examining table. The examiner places his hand under the calf and slowly flexes the hip, the leg being kept straight. The examiner's other hand is on the posterior surface of the thigh, to palpate the hamstring muscles. This movement (Fig. 3) stretches these muscles and will bring out any latent spasticity. The spasm can be palpated, is painful, and limits the motion. If the normal muscle is stretched

TABLE I

Lesion	may be associated with spasm of:
Sprain of the lumbar spine	Lumbar muscles Hamstring muscles
Sprain of the sacro-iliac area	Lumbar muscles Hamstring muscles Psoas magnus muscles
Sprain of the hip	Psoas magnus muscle
Sciatic neuritis	Hamstring muscles
Ischiogluteal bursitis	none
Coccygodynia	none

to its physiological limit at ninety degree flexion of the straight leg, it follows that a spastic muscle will permit less than this normal range of motion. The limitation and pain will be relieved on flexion of the knee for the reasons discussed in the section on "Mechanics of Normal Motion." The *difference* in the range of motion with the knee flexed and with the knee extended must be more marked when the hamstring muscles are spastic. All other motions that are ordinarily limited when the normal hamstring muscles have been stretched to a maximum will be much more limited when the muscle is spastic. Thus, in the standing position, flexion of the trunk, in so far as pelvic rotation is a factor, will be limited and elicit pain in the dorsum of the thigh. Complete flexion is possible when the knees are bent.

In the sitting position, because the knees are at right angles, there is no limitation in flexion of the trunk. The difference between the range of trunk flexion in standing and in sitting is striking.

A summary of the occurrence and symptoms of muscle spasm may be helpful at this point. In the left hand column of Table I, the common lower trunk injuries are listed and on the right the muscles which may be spastic during the acute stages of each lesion. In Table II the motions are listed which are limited and painful in the presence of spasm of each of the three muscle groups.

TABLE II

Motion which is limited and painful in spasm of	Hamstring m.	Lumbar m.	Psoas magnus m.
Standing			
Flexion of trunk			
A. Lumbar spine	o	++++	o
B. Pelvis	++++	+	o
Sitting			
Flexion of trunk			
A. Lumbar spine	o	++++	o
B. Pelvis	++	+	o
Lying			
Flexion of thigh, knee straight	++++	+++	o
Flexion of thigh, knee bent	++	+	
Flexion of both thighs, knees straight	++	++++	o
Flexion of both thighs, knees bent	+	++	
External rotation of the semiflexed thigh	o	o	++++

POST-TRAUMATIC SCIATIC NEURITIS (LEFT)

Inspection and palpation. Tilting of the spine is less common than in sacro-iliac conditions; when present it is to the left. Lumbar scoliosis, first described by Charcot and Babinski (1), is found in 80 per cent of cases; the convexity is to the right (contralateral scoliosis). The patient tends to keep the leg everted, the knee bent, and the foot extended. In severe cases the hip is flexed as little as possible. The standing position is more comfortable than sitting.

Tenderness of the nerve trunk may be present but is absent in the majority of cases. What is usually mistaken for nerve trunk tenderness is tenderness of the hamstring muscles and muscles of the calf. Atrophy of the thigh and calf muscles is an early and reliable sign. The circumference of the thigh and calf should be compared with the other side.

Determination of pain producing motions. When the patient lies flat on the table the sciatic nerve pursues an almost straight course from its lumbosacral origin to the internal malleolus, underneath which it turns forward to pass to the toes as the plantar nerve. It has been demonstrated on the cadaver (5) that the painful motions in sciatic neuritis are those which produce tension on the nerve roots by stretching the sciatic nerve trunk.

When the patient is on his back, the examiner flexes the left leg at the hip (Fig. 7). Pain is elicited, which may be referred anywhere along the course of the nerve. Pull on the nerve by this motion is due to angulation of the nerve at the hip. The main stress is due to pull on the nerve from below the point of this angulation. The mechanics of the main stress is analogous to the mechanics of pull on the hamstring muscles by this motion. On flexion the leg revolves about a center (the acetabulum) which is anterior to the center of motion of the sciatic nerve.. The tension on the nerve is immediately and almost completely relieved by flexion of the knee joint. On passive flexion of the thigh, with the knee

bent, little or no pain is elicited because the slack of the nerve by knee flexion is usually more than sufficient to compensate for the pull of the angulation at the hip joint. On passive flexion of the right (unaffected) leg, no pain is elicited.

Again passively flexing the left leg to the point at which pain is first elicited, the examiner dorsiflexes the ankle joint. Acute pain is produced in the course of the sciatic nerve. Dorsiflexion of the ankle joint can have no effect on any of the tissues of the lower trunk except the sciatic nerve. The angle which the nerve turns below the malleolus is increased on dorsiflexion of the ankle joint, thus causing a pull on the nerve trunk. Pain on this motion is one of the few signs which is characteristic of a single lower trunk injury. It was first described by Roussy.

When the straight leg is flexed at the hip, abduction and inward rotation relieve the tension on the sciatic nerve, because in this position the nerve course is more nearly a straight line. With abduction and inward rotation, therefore, greater range of motion without pain is possible. Adduction and outward rotation have the opposite effect. Flexion of the trunk is painful. That part of the flexion due to motion of the lumbar spine is free, but forward rotation of the pelvis elicits pain in sciatic neuritis, as the effect on the sciatic nerve is exactly the same as in passive flexion of the straight leg. In sitting, trunk flexion elicits little pain; this motion is comparable to flexion of the thigh with the knee bent.

The posture described under inspection is the position which causes the least pull on the nerve roots—namely eversion at the hip, flexion at the knee, and extension at the ankle joint.

Other physicial signs. Physical signs characteristic of nerve lesions may be found, such as absent tendo-achilli reflex, first described by von Sternberg. Areas of anæsthesia may be present. Because they so clearly indicate a nerve lesion as distinguished from lesions of other tissues involved in lower trunk injuries, they are of great importance when present. In some cases they are transient or are never present. In other cases they persist long after the pain and disability have disappeared. The pain on specific motion is more reliable than other signs as an index of the disability caused by neuritis.

Hamstring muscle spasm and sciatic neuritis. Sciatic neuritis in the acute stage is often associated with hamstring muscle spasm. The motions painful in the two conditions have many similarities, and the pain in both conditions is referred to the dorsum of the thigh. But hamstring spasm may be due to sacro-iliac and occasionally to lumbar spasm. The spastic muscles always should be palpated before a diagnosis is made. The pain on dorsiflexion of the ankle joint sometimes present in neuritis is a valuable sign. It is never found if the hamstring spasm is due to any lesion other than neuritis. Tenderness of the trunk of the nerve is helpful, but not often found. Most important is the search for signs of the other lesions producing hamstring spasm. The commonest mistake in the diagnosis of lower trunk injuries is to diagnose as sciatic neuritis the hamstring spasm due to sacro-iliac or lumbar strain.

Malingering and exaggeration. Backache is a frequent complaint in compensation cases, and in view of the financial benefits involved, the possibility of malingering must always be considered. A distinction must be made between malingering and exaggeration. When a patient has a long continued back pain and has been told by several physicians—"nothing to be seen, the ache will pass off"—it is not surprising that he grows apprehensive and exaggerates his symptoms when the pain persists. If the physician knows the physical signs it is not difficult to find inconsistencies which are the hall-mark of malingering. There is no single sign which proves malingering. It is the inconsistency of the signs on which that diagnosis is based.

A patient may allege inability to flex the trunk more than a few degrees on standing. When on his back it is found that thigh flexion and pelvis rotation are normal. The findings in the two positions are inconsistent. Even though he had a poker spine he could bend the trunk to a fair extent with normal motion at the hips. He is definitely malingering. When a patient alleges more marked restric-

tion and pain on flexion of the thigh with knee bent, than in straight leg raising, we know we are dealing with malingering.

I have seen the following signs erroneously described as signs of malingering:

1. With the patient prone on the table, he alleges pain on flexion of the knee. Comment: the flexion of the knee in this position, by pull on the rectus femoris, produces a forward rotation of the pelvis. It may, therefore, produce pain in the sacro-iliac or lumbar sprain, but never in sciatic neuritis or ischiogluteal bursitis.

2. A patient who keeps his lumbar spine rigid in flexion of the trunk, is placed supine on the table; on dorsiflexion of the two thighs simultaneously, it is found that the lumbar curve can be changed without pain. Comment: when the pain is due to strain on a muscle which has latent spasticity, the pain may be present in active flexion of the trunk when standing, and absent with passive motion of the spine when lying down. Sir John Collie describes this as a sign of malingering. It is occasionally found with lumbar muscle spasm and is then no indication of malingering. Failure to flex the spine is not the same as failure to flex the trunk.

3. The presence of exquisite superficial tenderness. Comment: the fact is that exquisite superficial tenderness may be present in the area of any spastic muscle.

SUMMARY

1. Each lower trunk lesion produces pain in a definite combination of motions which is characteristic of that lesion. Supposedly pathognomonic signs are misleading.

2. The physiological principles which account for the elicitation of pain by specific test motions have been described.

3. Each lesion may produce spasm in certain muscles and does not produce spasm of other muscles.

4. The various signs bear definite relationship to one another. By inconsistencies in these relationships, the diagnosis of malingering can be made.

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THE ENERGY BACKGROUND OF THE GENESIS OF GALL STONES AND OF THE PREVENTION OF IMMEDIATE POSTOPERATIVE SHOCK AND OF LATER DIGESTIVE DISTURBANCES[1]

GEORGE CRILE, M.D., F.A.C.S., CLEVELAND, OHIO
Cleveland Clinic

THE conceptions of classical physics were profoundly altered by the researches which followed the discovery of the X-ray. The conceptions of classical physiology and biology are now undergoing a profound alteration as the result of researches into the radiant and electric energy of protoplasm.

In Europe, following the work of Gurwitsch and others, these new conceptions regarding the rôle of electricity and radiation in the activities of protoplasm have advanced so far that the International Society of Electro-Radiobiology has been formed with an official journal *Radiobiologie*, the first number of which appeared in April, 1932.

If we are correct in our assumption—and this assumption is based on experimental evidence—that the organisms of animals and of man are operated by radiant and electric energy and that a mechanism has been set up to render this energy available for adaptive purposes, then on this basis we should expect variations in the relative sizes of the energy units of this mechanism—the brain, thyroid and adrenal sympathetic system, according to the adaptive needs of the species, and according to the varying demands of different age periods.

One of the characteristics which separates man from the other mammalia is his incessant activity, especially mental and emotional. Wild and domestic animals secure food, procreate, play, and sleep. Primitive or uncivilized man is relatively inactive. Civilized man, however, works all day and worries at night. The level of energy expenditure is one of the measures by which we may estimate the level of civilization. Therefore, one would draw the inference that the evolution of man involved the brain and also the gland that governs the rate of metabolism, namely, the thyroid gland.

With a normal thyroid there is normal brain activity. With a deficient thyroid there is diminished brain activity. With excessive thyroid activity there is excessive brain activity. The thyroid hormone maintains the level of activity of the brain. One would therefore expect the relative sizes of the thyroid, the adrenal sympathetic system, and the brain to vary with the energy characteristics of animals; that is, one would expect that the size of the thyroid would increase in proportion to the rate and constancy of demands for energy production, while the size and intricacy of the adrenal sympathetic system, on the other hand, would vary with the need for sudden use of that energy (Fig. 1). It would follow that the size of the thyroid in relation to body weight would be greater in man than in other mammalia, and that the adrenal glands, as Cannon has shown, being the organs that govern the outbursts of energy, would be larger in relation to body weight in the animals that depend on a rushing attack or a swift escape.

Moreover, one would suppose that the nerves and ganglia which are associated with the adrenals would be much more abundant and their arrangement more complicated in animals in the energy group such as the tiger, than in animals in the protected group such as the alligator.

Studies in the comparative anatomy of the brain, thyroid, and adrenal sympathetic system have shown that these assumptions are well founded, in particular as regards the intricacy and size of the adrenal sympathetic system which is large and complicated in the lion and tiger (Fig. 2), less so in man whose frontal lobe, however, is most highly developed (Fig. 3), while both the brain and the adrenal sympathetic system are small and uncomplicated in the crocodile and in the alligator (Fig. 4).

[1]Read at Temple University School of Medicine (on the occasion of the presentation of a portrait of Dr. W. Wayne Babcock to the School by the third and fourth year classes), Philadelphia, Pennsylvania, May 2, 1934.

If the brain, the thyroid gland, and the adrenal sympathetic system are thus linked together, it would follow that in a state of abnormal activity, or what we may term pathological physiology of this great system, the whole organism would be affected, with the resultant production in one individual of hyperthyroidism, in another of diabetes, in another of peptic ulcer, in another of gall stones. None of these originates within the affected organ. The thyroid gland does not originate hyperplasia; hyperplasia is imposed upon the thyroid gland. The stomach does not originate a peptic ulcer; the condition within the stomach which produces an ulcer is imposed upon the stomach by influences outside itself. The pancreas does not originate diabetes; diabetes is imposed upon the pancreas, and so it is with gall stones, their origin is not within the gall bladder, nor within the liver, but the condition which produces gall stones is imposed upon the liver by influences outside itself.

That emotion, physical exertion—any excess transformation of energy—affects the whole energy system is well known, as has been shown by Cannon and ourselves. That such hyperactivity of the adrenal sympathetic system affects the liver has been demonstrated by our histological and biophysical researches.

Twenty-four years ago we conducted an extensive histological research to discover what organs and tissues are affected by excess or diminished transformation of energy in the body. In this research a histological examination was made of every organ and tissue in animals which had been subjected to insomnia, to fear, to physical injury, to shock, to prolonged anæsthesia, to infection. In every case histological changes were observed in three organs and in three organs only, the brain, the liver, and the adrenals. The liver cells lost their differential stainability—they became swollen and œdematous; the cytoplasm was vacuolated; the nuclei were crenated and the cell membranes were irregular, the most marked changes occurring in the cells nearest the periphery of the lobules (Fig. 5). Identical changes were found in the liver cells of patients who had died from infection, puerperal eclampsia, hyperthyroidism, or in any

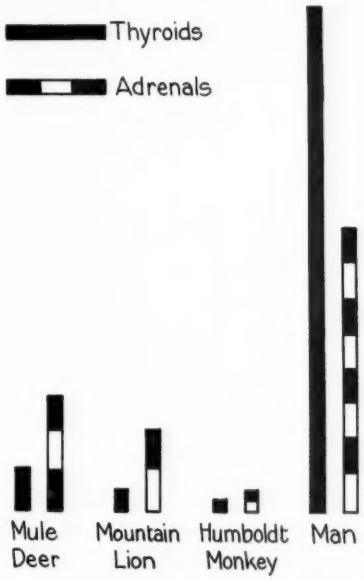


Fig. 1. Comparative sizes of the thyroid and adrenal glands (charted from actual weights).

case in which exhaustion was due to the protraction or intensity of the disease. Later biophysical researches disclosed that, under identical conditions, changes took place in the temperature and in the electric conductivity, electric capacity, and electric potential of the liver. These experimental facts confirm the clinical deduction that the change in the bile which leads to gall stones is not due to changes in the gall bladder, but to changes in the function of the liver cells.

It would follow that one would expect to find the highest incidence of gall stones among races and individuals in whom the energy system is peculiarly active. How is this assumption borne out by known facts regarding the incidence of gall stones? Gall stones are rarely seen in wild animals in which, although adequate for attack and defense, the adrenal sympathetic system is not subject to prolonged stresses and strains. Dr. Herbert Fox, Pathologist at the Philadelphia Zoological Gardens, has reported the finding of gall stones in but 14 of 5,365 autopsies on wild animals in captivity. Gall stones are less common in the lower races of man than in civilized races, and

among civilized races gall stones are less common among peoples who have a negative philosophy. Gall stones are most commonly found in Americans and Europeans; less commonly in Asiatics. While gall-stone disease is relatively common among Americans and Europeans, it is most common among people of the Hebrew and Latin races.

In other words, gall stones occur most frequently in the active, aggressive section of the human family, their incidence corresponding roughly to the distribution of hyperthyroidism, peptic ulcer, neurocirculatory asthenia, diabetes, highly emotional temperaments, and high scholastic, high executive, and administrative ability.

Another significant fact is that among humans the incidence of gall stones is higher among females than among males, especially among women who have given birth to children.

In pregnancy there is increased metabolism with hypertrophy of the thyroid and of the adrenal glands; there is a gain in weight; nervousness and palpitation are present; the organism is stepped up as in the expression of the emotions, in defense against infection, in excessive and fatigue-producing work. Dr. E. L. Walsh, George W. Belcher Research Fellow of the Cleveland Clinic Foundation, states that hypercholesterinæmia is present in pregnancy. Therefore, since the liver cells are the only source of the bile, when they are interfered with so profoundly as in pregnancy, the concentration of the bile is changed, and therefore the formation of gall stones is facilitated. But pregnancy itself is not the only reason why a married woman with children is more commonly affected by gall stones than are men or non-childbearing women. The married woman has the children and the household and a husband on her hands as well. She is peculiarly subject to restraints and worries and anxieties and emotional strains, all of which involve the adrenal sympathetic system and in consequence, as we have demonstrated, disturb the function of the cells of the liver. To that extent, the childbearing woman is more prone to have gall stones than is her husband or the woman who has borne no children.

Any theory that is proposed to account for gall stones must account for all these facts. Our thesis is that the chemical and physical basis of gall stones is laid by changes in the secretion of the biologically active liver cells and not by changes in the sac that holds and concentrates the bile and performs no other function.

Let us consider the means by which the bile carries in solution such large quantities of cholesterol and bile pigment without forming gall stones in every human gall bladder. Dr. A. C. Ivy and Dr. E. L. Walsh and others have demonstrated clearly, by their studies of the effect upon human gall stones of placing them in the gall bladder of a dog, and by experiments on the solution of gall stones *in vitro*, that the outstanding factors that govern the maintenance of the cholesterol and pigment in solution in the gall bladder and ducts are the bile salts and the fatty acids. We may conclude that when these are present in sufficient concentration there apparently will be no formation of gall stones. Therefore, when gall stones form, they form because there is an insufficient concentration of bile salts and fatty acids, or perhaps an excess of cholesterol.

We know that in highly developed, high-strung, emotional, active people and in childbearing women the adrenal sympathetic system is hyperactive. We know that hyperactivity of the adrenal sympathetic system profoundly affects the cells of the liver. We know that when the liver cells are so altered, they will not secrete a normal quantity of bile salts and fatty acids.

A significant fact is that comparatively few of the individuals—animal or human—that have gall stones also have cholecystitis or gall-stone colic. Since 10 per cent of the inhabitants of Europe and America have gall stones, there should be approximately twelve millions of gall bladders which contain gall stones in the American population at this time, yet the total number of operations for gall stones and the total number of cases of gall-stone colic are relatively small.

We must offer evidence, therefore, that gall stones are not produced primarily by infection of the gall bladder, as is the common belief. If, in a hundred-thousand cases gall

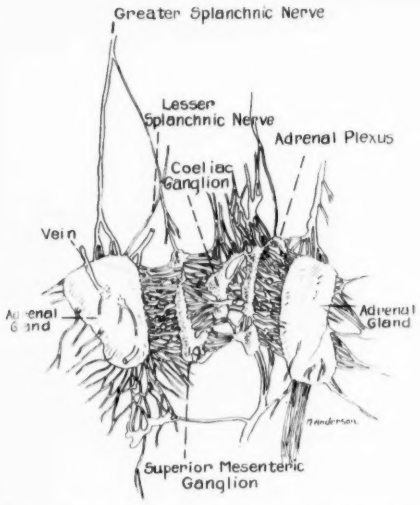


Fig. 2. The adrenal-sympathetic system of the tiger.

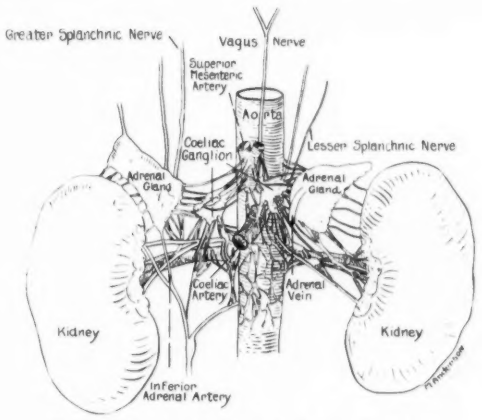


Fig. 3. The adrenal-sympathetic system of man.

stones were taken from the gall bladder and replaced by any other foreign body of the same size and weight, one might expect the incidence of infection and abscess to be approximately the same as in the case of gall stones in nature. In either case, the infection might be due to the local irritation from the foreign body or from the gall stones. The question raised is whether the infection is not the *result* rather than the *cause* of gall stones. That this is the case is attested by the fact that there are at this moment, in the United States, approximately twelve million people who have gall stones in their gall bladders and do not know it. Moreover, and this is an even more important fact, relatively few patients have an infected gall bladder or an abscess in the gall bladder without gall stones.

It is readily to be seen that when there is an interference by gall stones or infection with the free flow of bile in and out of the gall bladder, there results an immediate interference with the concentration of the bile salts and fatty acids, and to that extent the further formation of gall stones is promoted by the diminished concentration of the solvent bile salts and fatty acids. Thus, an infection of the gall bladder, by interfering with the concentration of the bile, would probably promote the formation of gall stones. We believe, however, that local infection is more commonly secondary, just as, in many cases of hyperthyroidism, of peptic ulcer, of Raynaud's disease, of intestinal obstruction, there are many secondary effects which arise from the primary cause. Therefore, we may think of an infection of the gall bladder as being a complication of stones rather than of stones being a complication of the infection.

In this connection, it is pertinent to recall that there was a time, following the work of Halsted, when it was believed by many pathologists and surgeons that gall stones were commonly due to the presence of typhoid bacilli in the gall bladder. It is now about 30 years since typhoid fever has been practically eliminated as a disease, and therefore, if this

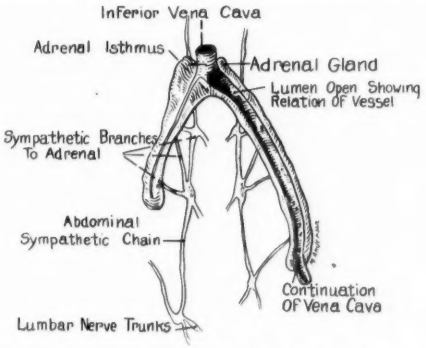
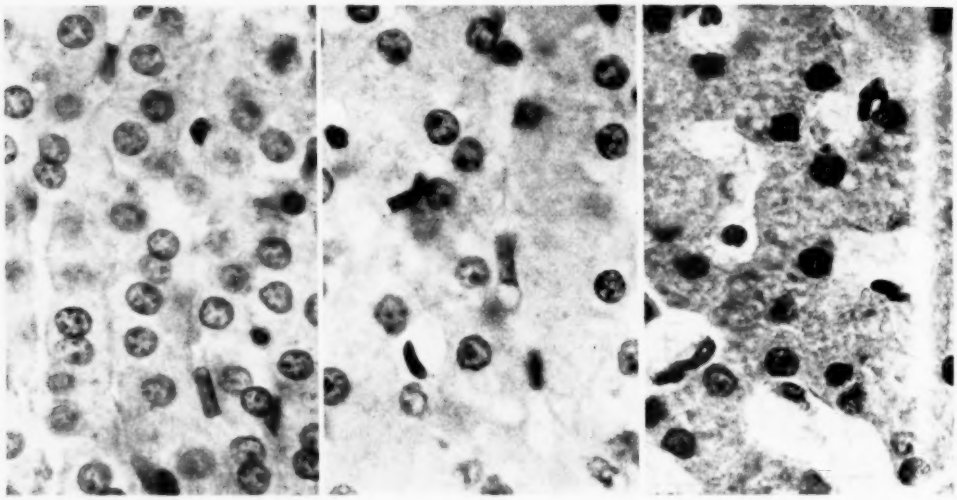


Fig. 4. The adrenal-sympathetic system of the alligator.



A B C

Fig. 5. Effect upon the liver cells of ether anæsthesia, B, and of adrenalin, C. A, Section from the liver of a normal dog. B, Section from the liver of a dog which had been subjected to continuous ether anæsthesia for 4 hours. Note the loss of cells and vacuolated spaces. C, Section from the liver of a dog which had been subjected to repeated doses of adrenalin. Note the loss, misplacement, and crenation of cells and vacuolated spaces.

assumption were true, the incidence of gall stones should be reduced by about 50 per cent. This is definitely not true—typhoid has disappeared; gall stones have remained. If gall stones are produced by the pyogenic infections, the greatest incidence of gall stones should occur in early life in the age of the highest incidence of infectious diseases, in the period of the highest incidence of appendicitis, of tonsillitis, of tuberculosis, of osteomyelitis. These infections occur early in life—gall stones later.

Finally, if it were a fact that infections have an affinity for the gall bladder, then acute gall-bladder infection should be as common as tonsillitis, for the liver absorbs every kind of toxin and bacteria from the intestine, but it does not commonly become infected. The duodenum, the stomach, and the intestines are far more exposed to contamination than are the gall bladder and ducts, but they are rarely infected because they have a natural resistance; the gall bladder and ducts have a natural resistance and they too rarely have an acute infection except in the presence of stones which irritate them. If the stomach and duodenum are likewise irritated or damaged they too may become infected. It is indeed difficult to establish even a probability that the genesis of gall stones is due to infection, except so far as the infection may have altered the liver cells.

We may conclude, therefore, that gall stones are due to an often repeated or continuous interference with the quantity and quality of the secretion of the liver cells, which results in a deficiency of bile salts and fatty acids, and therefore produces an abnormal quality of bile.

We can see, therefore, why the more emotional the individual or the race the more common the gall stones; how excessive fatigue or prolonged or repeated infections or pregnancy may predispose an individual to the formation of gall stones; why the Jews and the Latins show a higher incidence of gall stones than do such races as the philosophical Nordics or the Chinese and the Hindus with their peculiar diet and negative philosophies of Confucianism or Brahmanism, or any human beings who have rationalized their lives and freed them from their stresses. What is there to change the liver cells in the primitive man who needs only to pluck an orange,

or dig up a root, and then can sleep and loaf the rest of the day?

It is the adrenal sympathetic system acting upon the liver, not the gall bladder and bile ducts, which provides the fundamental cause of gall stones. It is not local infection, not pathological morphology, but pathological physiology which lays the background for the formation of gall stones.

We have an additional line of very definite evidence to offer. Dr. Walsh has stated that in an experimental animal in which a biliary fistula has been produced, in the presence of infection, the bile is not secreted in the usual amount and in normal concentration until several days after the animal has been subjected to a surgical operation, and that any laboratory animal that is in ill health shows a diminution of the amount of bile secreted and in its concentration.

If there is any one fact that the experienced surgeon knows clearly regarding the sequence after operation on the gall bladder, it is that a great change takes place in the quality and the quantity of the bile as the result of the surgical operation. At the present time, when surgeons are commonly removing the gall bladder, there is little opportunity for observing the flow of bile as in former periods when we removed the stones and drained the gall bladder. Then there was a perfect opportunity to note the effect that the anæsthetic, the shock, infection, etc., had upon the quality and the flow of bile, in other words, upon the function of the liver cells. Every surgeon who remembers those days or who at the present time has observed the character of the bile in serious cases, as in cases of jaundice in which the gall bladder is drained, has noted that the liver cells were not able to secrete bile for a period of time, perhaps not until 24, 48, or even 72 hours, after the operation, and he will remember his relief when normal bile again began to flow.

Every surgeon probably has performed apparently successful operations on the biliary passages of patients whose liver function was depressed. At the close of the operation the prognosis would seem promising, but the following postoperative picture would soon begin to develop: The patient would become increasingly apathetic; his energy would be slowly depleted; though tired and weak he would not be able to sleep; the tongue would become increasingly dry; the pulse would gradually rise and its volume steadily decrease; the bile would be suppressed; nourishment would be refused; the patient's demeanor would be cheerless and despondent. The patient would seem to be sinking steadily in the quicksand of some fundamental failure of normal metabolism. No means would suffice to change the course of events. The patient would either recover gradually or else would become first delirious and then unconscious until death.

This is a familiar picture after operations on patients exhausted by chronic empyema of the gall bladder or by long continued infections from common duct stones. It is a familiar picture after operations on patients exhausted by prolonged or by sudden intense psychic strain or by some severe chronic infection. The temperature and pulse of these patients may be normal and if the prognosis were based on these factors alone the greatest errors would be made.

We now know that this picture was due to the effect of the surgical trauma, the anæsthetic, and the psychic influences which attended the operation. All of these affect the adrenal sympathetic system and through the sympathetic system profoundly affect the liver cells.

It has been established by laboratory experimentation that the liver performs its function in part through direct nerve stimulation over the sympathetic system and in part through hormone action. The nerve supply of the liver is derived from the sympathetic and parasympathetic systems. These nerve fibers pass along the blood vessels and the common duct. In addition to the nerve supply to the liver as a whole, each separate liver cell has its own nerve supply; not only is its outer border supplied, but a filament of nerve tissue penetrates each separate cell (Fig. 6). One would expect to find that an organ, every unit of whose structure is thus wired, would have a close relationship with the great functions of the body.

Anyone who has operated upon the thyroid gland will remember that even an approach

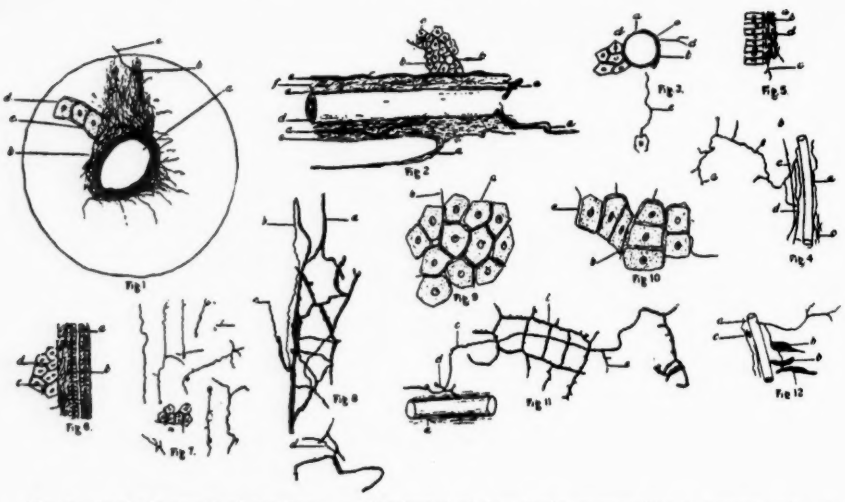


Fig. 6. Intrinsic nerves of the liver. 1. Meshwork of fine fibrils surrounding a central vein. 2, Drawing from a section not far from the transverse fissure of the liver. 3, Central vein of a lobule with surrounding nerve fibers. 4, Nerves and their branches surrounding a portion of a hepatic artery. 5, Portion of a biliary canal showing at C nerve fiber with a forked termination at *d*. 6, Biliary duct with nerve fibers and intercellular branchings. 7, Varieties of nerves and terminations. 8, Portion of a coarse biliary plexus along the margin of an interlobular space. 9, Portion of an intercellular hepatic plexus. 10, Portion of the terminal distribution of a single nerve fiber. 11, Portion of a biliary plexus. 12, Neural enlargements adjacent to a portal vein in an interlobular space. (From Berkley: Studies in the Histology of the Liver. The Johns Hopkins Hospital Reports, 1895, iv, 231.)

to the recurrent nerve—merely touching it, or producing the slightest traction on it—causes at once a great change in the conductivity of that nerve, which is expressed by a change in the voice. The reason is that this nerve is poorly protected as are all nerves which, being deep-seated, have always been phylogenetically protected from injury. If it were possible for injury of the liver cells to be demonstrated by crying out, as is injury of the laryngeal nerve, the demonstration would be dramatic indeed.

Since, as our experimental researches have shown, physical trauma and inhalation anæsthesia affect the liver cells, it is obvious that, for the protection of liver function, minimum trauma must be inflicted and that the anæsthesia must not be carried beyond the minimum degree necessary to secure the required relaxation, principal dependence being placed upon local, regional, and splanchnic anæsthesia.

After leaving the hospital, many patients complain of a group of distressing symptoms: (1) lack of vigor and endurance, (2) gas pains, (3) indigestion. These symptoms may continue for many months—even for a year or more. It would seem that their genesis may now be explained and these sequelæ prevented, because, as we believe, the symptoms are due to injury of the sympathetic nerves which are so richly supplied in the precise area of the operative attack. These nerves are as delicate as the spinal cord. What if the neurosurgeon should pack the brain, the medulla, the spinal cord to stop bleeding or retract them with force to make up for the relaxation a proper anæsthetic would give! Not only does the surgeon cause a profound interference with the innervation of the liver, but also with the whole adrenal sympathetic system—with the innervation of the pancreas, the adrenal glands, the entire gastro-intestinal tract. If in addition to such injury his sensitive, exquisitely delicate telephone switchboard of the sympathetic system is harmed enough to cause scar tissue to form, then his system will cause the above mentioned sy[illegible]p-

toms to continue for months or years, just as in the case of injury of the recurrent nerve in a roughly conducted thyroidectomy with the formation of scar tissue.

The indigestion and lassitude following operations on the gall bladder and ducts is the surgeon's disease. With this conception in mind, I so plan and execute the operation that the network of the sympathetic is only slightly disturbed, and as a result these distressing postoperative symptoms are no more than those which follow a pelvic operation.

From these considerations it would appear that the genesis of gall stones and the prevention of postoperative shock and of later digestive disturbances are related to the energy system which is the mechanism through which the radiant and electric energy operating the organism is principally expressed.

SUMMARY

1. A transformation in regard to our conceptions of the energy whereby living organisms are operated is now in progress and will inevitably affect biology and medicine.

2. There is nothing more definitely established than the fact that the injection of adrenalin, the expression of an emotion, foreign proteins, an infection, a great exertion, a physical injury, pregnancy, all produce an increased activity of the energy system of the organism, and the liver is linked indissolubly with that energy system. In other words, all the factors that cause an increased output of adrenalin in the experimental laboratory have the power of changing, of modifying, of interfering with the cells of the liver, and, in consequence, with their production of normal bile.

3. This change in the liver cells is the result of hyperactivity of the adrenal sympathetic system.

4. Change in the cells of the liver in turn predetermines a change in the bile content; change in the bile content, as shown by the researches cited, leads to the formation of gall stones.

5. Immediate shock and postoperative indigestion and pain are almost completely prevented by avoiding injury of the exquisitely sensitive sympathetic nerves by avoiding the formation of scar tissue which involves the rich and intricate network of the sympathetic nervous system.

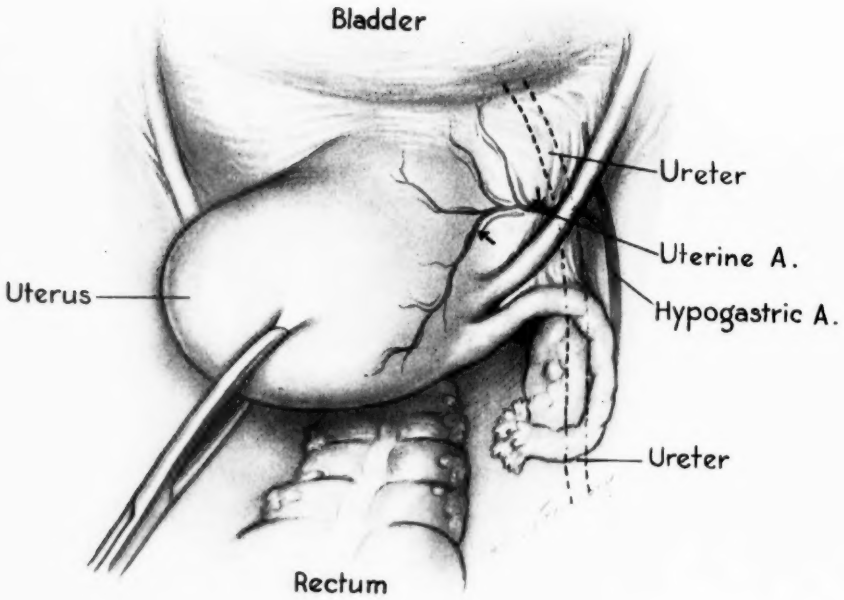


Fig 9. Step 6.

Total Abdominal Hysterectomy.—Lilian K. P. Farrar

CLINICAL SURGERY

FROM THE CLINIC OF THE WOMAN'S HOSPITAL, NEW YORK

TOTAL ABDOMINAL HYSTERECTOMY

ANATOMY AND TECHNIQUE[1]

LILIAN K. P. FARRAR, A.B., M.D., F.A.C.S., NEW YORK, NEW YORK

THE operation of hysterectomy has been a matter of evolution over a period of approximately 50 years. Probably the greatest impetus and contribution to the technique was given by Dr. Lewis A. Stimson, a founder and the first professor of surgery in Cornell University Medical College. On January 9, 1889, Dr. Stimson described before the New York Surgical Society the technique of two total abdominal hysterectomies which he had just performed. In these two operations Dr. Stimson had applied ligatures "to the uterine arteries alone underneath the peritoneum" with the object of preventing, first, hæmorrhage from the retraction of the blood vessels, second, the sloughing of the pedicle. In this meeting Dr. Stimson asked the opinion of his fellow members of the relative value of total or subtotal hysterectomy, saying that he himself believed the total to be the better procedure. In July of that year Dr. Stimson published his paper entitled "On Some Modifications in the Technique of Abdominal Surgery Limiting the Use of the Ligature *en Masse*" and stated that if the cervix was left *in situ* it should be covered with a flap of peritoneum, thus solving the problem of the treatment of the pedicle or stump which had so long been a stumbling block in hysterectomy. The value of this and the isolation before ligation of the uterine artery was quickly recognized. These two procedures have revolutionized the operation of supravaginal hysterectomy—but total hysterectomy, which Dr. Stimson advocated, has not been, at least in this country, generally accepted for benign condition of the uterus.

The ease with which the supravaginal method may be carried out and the brilliant work of the leaders of the next decade have led to its great popularity in the United States—so much so that in Europe today the supravaginal hysterectomy is called "the American operation." In France Doyen, Wiart and Forgue, and in Germany and Austria Bumm, Doederlein and Weibel, and before the latter Wertheim, have all advocated the total operation for non-malignant conditions necessitating hysterectomy. In England Mr. Herbert Spencer has for years practiced total hysterectomy for benign conditions of the uterus and has published statistics for all of his cases. The wisdom of this routine practice lies in the knowledge that cancer is not infrequently associated with fibroids of the fundus and that cancer of the cervix has been reported by numerous surgeons to have developed later in the cervical stump. The incidence of cancer occurring with fibroids is probably about 5 to 8 per cent. Munro-Kerr found in his cases 5 per cent malignancy in 200 fibroid tumors of the uterus, and quotes Ellice McDonald as finding 5 per cent malignancy in 700 myomata uteri. Polak studied 900 fibroids and found cancer undiagnosed and present in 2 per cent. The difficulty of curetting out all tissue from a uterine cavity distorted by myomata might be a reason for not diagnosing even a suspected case of malignancy, and a second reason is that sarcoma rarely begins in the endometrium. An additional reason for the removal of the cervix together with the fundus was first given in 1896 by Chrobak, of the University Clinic of Vienna, who reported 3 cases of cancer that had developed in the stump of a cervix several years after supravaginal hysterectomy had been done. In 1910 Dr. John Osborn Polak collected from American literature alone 256 cases of carcinoma occurring in the cervix one year or longer after a supravaginal hysterectomy had been done; and from all literature 900 cases of carcinoma occurring in the cervix thus left—Masson reported 29 cases of carcinoma in the cervix after supravaginal hysterectomy seen in 5 years at the Mayo clinic.

In the Woman's Hospital the incidence of carcinoma developing in the cervical stump one or more years after a supravaginal hysterectomy is 7 per cent of all patients who come to the clinic for treatment of carcinoma of the cervix. Spencer

[1] Read before the American Gynecological Society in White Sulphur Springs, Virginia, May, 1934.

quotes Peham and Amreich that carcinoma is 27 times more frequent in the cervical stump than in the cervix of women who have not had a subtotal hysterectomy. If this be so is it not probable that we are making tissue favorable to the development of cancer when we leave a cervix in doing a hysterectomy. Tissue that has been devitalized by any means such as by burning, cauterization, cutting off the blood supply, makes that tissue more liable to become infected.

Leucorrhœa is a frequent symptom after supravaginal hysterectomy in women who have never had a vaginal discharge before. Leucorrhœa results from the degenerative changes that follow in the mucous glands of the cervix. A chronically diseased cervix stimulates tissue reaction and cell proliferation and thus paves the way to the development of cancer. Leucorrhœa and backache are such common symptoms after the subtotal operation that many surgeons cone out the cervical canal from above at the time of the amputation of the fundus, or cauterize the cervical canal from below immediately preceding the abdominal part of the operation. This latter treatment, however, may be a source of severe infection as the slough lies so close to the abdominal wound. The coning out of the cervical tissue may or may not cure the leucorrhœa and may prevent the development of cancer, depending upon how thoroughly the base of the glands are destroyed; but Peterson has shown that only one-third of the carcinoma of the cervix begins in the cervical canal while two-thirds are of epithelial origin and begin in the portio of the cervix, which is not destroyed by the coning out or cauterization of the canal. Years ago Doederlein was asked what he considered the best treatment of the cervix to be. He replied: "Die beste Stumpbehandlung der Zervix ist eben deren ganzliche Wegnahme." This seems to be true today, for as Dr. W. J. Mayo has written "We must look upon local lesions as an invitation to cancer without regard to just what the actual cause of cancer may be . . . . Leaving the cervix leaves an average cancer liability." And again Dr. Mayo says "I believe that total hysterectomy is a wise procedure if it can be done safely and usually it can."

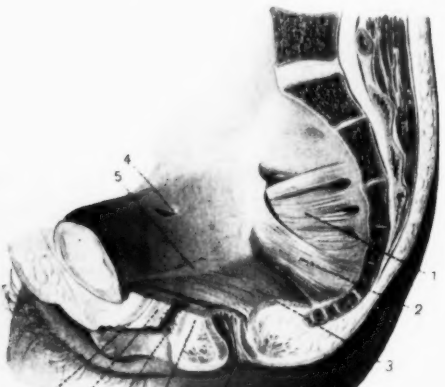


Fig. 1. The connective tissue takes its origin from the arcus tendineus. *1*, M. piriformis; *2*, M. coccygeus; *3*, M. levator ani; *4*, obturator canal; *5*, arcus tendineus m. levator ani; *6*, urethra; *7*, urethrovaginal septum; *8*, vagina; *9*, rectovaginal septum; *10*, rectum.

The value of the total hysterectomy and the development of a simple technique was shown in 1917 by Dr. F. F. Baldwin of Columbus, Ohio. Dr. Baldwin wrote: "It entirely removes the possibility of malignant changes in the retained cervix and more or less morbidity from inflammatory or degenerative changes which may be present or which may occur in the part that is left behind." A year or two later Polak of New York published his technique, demonstrating particularly the removal of the cervix. Worrall of New Zealand at this time presented his method of leaving a strip of the lateral walls of the uterus so as not to disturb the insertions of the ligaments and thus prevent prolapse. Lahey has much the same technique with especial guidance on cutting out the cervix. Obviously though, this strip of uterus should not be left if cancer is present in the fundus and not infrequently cancer may be present and not diagnosed. Kennedy of the Woman's Hospital utilizes the cervical ligaments to prevent prolapse as in the vaginal hysterectomy. Richardson of Baltimore, in 1929, presented an admirably planned technique with the attention directed especially to the prevention of hæmorrhage.

The higher mortality in the total hysterectomy is a deterrent to many surgeons. It may be desirable then to consider the mortality of the two operations. A fair statement is that of Diamant-Berger of the Hôtel Laboisiere who says "All surgeons perform daily the subtotal, how many on the other hand have the same experience with the total? Is it not true then that they reserve the total operation for the most seriously ill, the infected, or those suspected of cancer of the fundus? *Il est donc artificiel et illogique de comparer des choses qui ne sont au fond pas comparables même entre les memesmains.*" Certainly one cannot acquire the same skill in doing the less frequent operation as in the operation which one performs

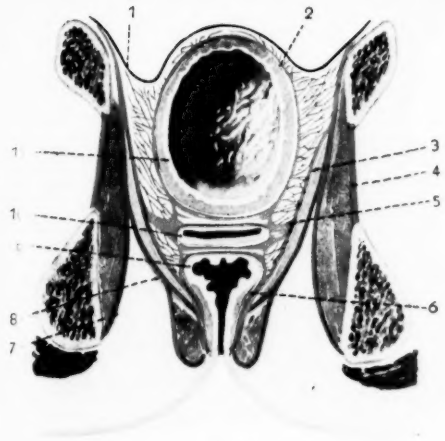


Fig. 2.

Fig 3.

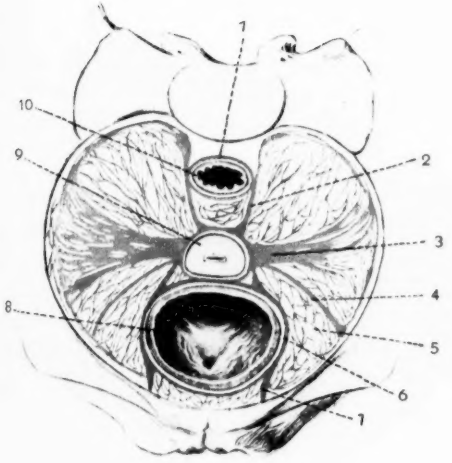


Fig. 4.

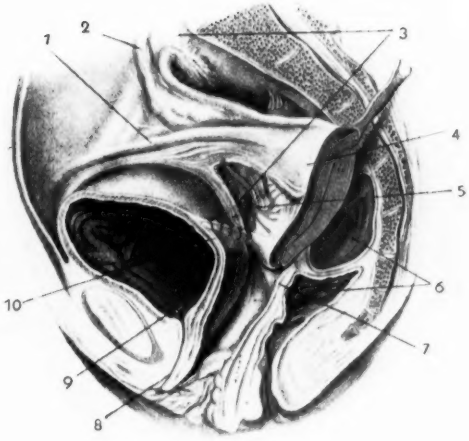


Fig. 5.

Fig. 2. The endopelvic fascia covers over the pelvic organs, separating one from another. *1*, Peritoneum; *2*, perivesical connective tissue; *3*, pelvic fascia; *4*, obturator membrane; *5*, endopelvic fascia; *6*, perirectal connective tissue; *7*, obturator fascia; *8*, deep perineal fascia; *9*, rectum; *10*, vagina; *11*, bladder.

Fig. 3. Septa between bladder and uterus and between uterus and rectum. *1*, Perirectal connective tissue; *2*, pelvic fascia; *3*, deep perineal fascia; *4*, perivesical connective tissue; *5*, perivaginal connective tissue.

Fig. 4. The endopelvic fascia radiates like an open fan from lateral wall of the uterus to the pelvis. *1*, Perirectal connective tissue; *2*, sacro-uterine ligament; *3*, cardinal ligament; *4*, ligamentum teres; *5*, loose connective tissue; *6*, perivesical connective tissue; *7*, pubovesical ligament; *8*, bladder; *9*, uterus; *10*, rectum.

Fig. 5. Dissection showing the blood supply of the uterus. *3*, Ureters; *4*, uterus; *5*, annular artery; *6*, rectum; *7*, posterior vaginal wall; *8*, vagina; *9*, sound; *10*, anterior wall of vagina.

TABLE I.—FIGURES OF READ AND BELL

	Subtotal hysterectomy	Mortality per cent	Total hysterectomy	Mortality per cent
Read and Bell	1739	2.1	605	3.1
Fullerton and Faulkner	609	4.4	1078	4.1
Weibel	not given	4.25	not given	3.55

TABLE II.—NELSON'S FIGURES

	Subtotal hysterectomy	Mortality per cent	Total hysterectomy	Mortality per cent
Mayo clinic (1926)	217	1.8	229	1.3
Ford Hospital	122	3.2	476	2.9
C. H. Mayo (1931)	3085	1.2	1588	1.8
Kennedy, W. T.			191	3.1

daily. Polak has estimated that the mortality of subtotal hysterectomy should be not over 1.5 per cent and the total not over 2 per cent. While many may consider these figures too low and the ratio between the two operations to be too small, one must bear in mind that to the mortality rate for the subtotal operation certain figures must be added to tell the whole story. When a cervical stump that has an unsuspected carcinoma is left or when carcinoma develops in it later the subsequent mortality or morbidity should be added to the primary mortality of the subtotal operation. Radium treatment for carcinoma in the cervical stump is more difficult and more liable to be followed by a vesical fistula, as the bladder is directly over the cervical canal. Radium burns of the intestine are not unusual if a supravaginal hysterectomy has been previously performed and intestinal adhesions have resulted. Treatment or amputation of an infected cervix left after the subtotal operation should also count against the incomplete operation. It may, however, be of interest to view the statistics of several hospitals (staffs) for both total and subtotal hysterectomies, and also the statistics of individual operators. Read and Bell give the figures shown in Table I for 14 surgeons in 10 years at the Chelsea Hospital in England. Nelson's figures are shown in Table II.

This makes 2,712 total hysterectomies done not only by individuals but by the staffs of general hospitals with a mortality less than 2 per cent.

The criticism may be made that the statistics given are for the total hysterectomy as done by specially trained groups and not by the great number of surgeons doing gynecology as part of general surgery, for as Dr. John G. Clark wrote several years ago in his article on the abdominal operation for cancer of the cervix: "The universal benefit of any medical or surgical measure cannot be great if its execution is so difficult as to render it highly hazardous except in the hands of a very few specialists." It is with the desire to help simplify the operation of total hysterectomy that I

TABLE III.—INDIVIDUAL SURGEONS—TOTAL HYSTERECTOMY FOR FIBROMYOMATA

	Total hysterectomy	Mortality per cent
Lockyer	195	1.54
Spencer	325	1.8
Ott	375	0
Richardson	not given	0

report this technique. The usual objections given to total hysterectomy are (1) more difficult technique, (2) danger of hæmorrhage, (3) danger of injuries to bladder or ureters, (4) infection from the vagina, (5) shortening of the vagina, or prolapse of the vault of the vagina. It may be well to consider these objections first.

The technique. A consideration of the anatomy of the tissues involved may help to a better understanding of the technique to be described. The complexity of the structures in the pelvis, and the multiplicity of terms used by different writers has led to much confusion as to the purpose of these structures. The best description today of the pelvic anatomy is, I believe, that written by Dr. Julius Tandler, professor of anatomy in the University Clinic in Vienna. Briefly summarized it is as follows: An analysis of the endopelvic fascia (called "endo"—within—the pelvis simply to distinguish it from the fascia that is outside, lining the walls of the pelvis) shows that

1. It is a true muscle fascia. The connective tissue takes its origin from the arcus tendineus on each side of the pelvis (Fig. 1) and extends to the mid pelvis to be inserted into the lateral walls of the uterus, cervix, and vagina. The underlying muscle is derived from the organs to which it is attached.

2. This endopelvic fascia covers over the pelvic organs, separating one organ from another, and from the serosa which lies above or around each organ (Fig. 2). It sends down septa between the bladder and uterus known as the septum vesico-vaginale and between the uterus and rectum, the septum recto-vaginale. (Fig. 3.)

3. In some places this endopelvic fascia is greatly strengthened and forms true ligaments but in other places it exists merely as an areola network the interstices of which are supplied with fat to fill in the spaces between organs. From above, the endopelvic fascia appears as a broad sheet of fascia. Anteriorly at the base of the bladder the fascia is much thickened and together with its muscle which is derived from the vagina and bladder it extends from the os pubis to the vagina and cervix as the ligamentum pubo-vesico-vaginale. Laterally on each side of the pelvis the endopelvic fascia is composed of thick bundles of connective tissue and smooth muscle and extends

from the pelvic wall, to be inserted into the lateral walls of the uterus and cervix. Looking from above the endopelvic fascia (Fig. 4) radiates like an open fan from the lateral wall of the uterus to the pelvis. In sagittal section it is three-cornered. The muscle is thickest at its insertion into the uterus but varies in amount, with the age and development of the individual. In pregnancy the muscle is greatly thickened and the blood supply increased to make a vascular muscle sling for the uterus. Later in life this muscle atrophies and the fat is absorbed. The nerves, lymphatics, and blood vessels supplying the uterus, cervix, and upper vagina lie in its depths. The whole forms the so called "parametrium." A variety of names have been given to this structure. It is known as the ligamentum transversum colli of Markenrodt, the ligamentum cardinale of Koch, the sustentaculum of Bonney, the upper pelvic floor of Polk, etc. (The anterior and posterior lamella of the peritoneum cover the parametrium, the fallopian tube and mesosalpinx, and include the ligamentum ovarii proprium and mesovarium to form the broad ligament.) At the anterior edge of the parametrium the endopelvic fascia continues on each side of the pelvis as a thick bundle of connective tissue and smooth muscle to form the so called ligamentum teres. At the posterior edge of the parametrium on each side it forms in similar way the ligamentum sacro-uterinum which unites with its fellow from the other side to surround the rectum. Between these different ligaments are spaces filled with areolar connective tissue and fat. This whole sheet of endopelvic fascia when seen from above might be likened to a cartwheel, the spokes of which are the ligaments joined at a central point, the hub, or uterus. When the hub, or uterus, is cut away the spokes, or ligaments, must be united at a new central point. This joining of ligaments ensures a long vagina without danger of prolapse.

2 and *3. Hæmorrhage, bladder, and ureters.* The blood supply of the body of the uterus, the cervix, and the upper vagina and bladder comes from the uterine artery which is derived on each side from the hypogastric artery. After the uterine artery has crossed the ureter and before it enters the uterus it gives off three or four branches. The branch nearest to the uterus (Fig. 5) is the annular artery which unites with the artery on the opposite side to supply the cervix. The second branch is the anterior vaginal artery which supplies the upper vagina and the third branch is the vesical which goes to the posterior wall of the bladder. The uterine veins follow the course of the uterine arteries. The venous return is carried

Fig. 6. Blood supply of the uterus after injection of the two uterine arteries. (From Deve, Théses d. l. Fac. de méd., Paris.)

up by the uterine veins to the hypogastric veins and on the right side to the inferior vena cava, on the left usually to the left renal vein. In 1892 Dr. William M. Polk demonstrated, before the American Gynecological Society, the anatomical arrangement of these vessels and advocated ligating the uterine artery median to the anterior vaginal branch. These arteries and the plexus of veins have been clearly shown by X-ray after injection (Fig. 6) of the blood vessels of the uterus. Faure and Wiart in France, Bonney in England, Richardson and Lahey in this country have stressed the importance of properly applied clamps to control the arterial bleeding and the venous oozing at the vaginal angles. Bonney says: "If the arteries have been properly isolated and the clamp has been applied at a point *just distal* to the *origin of the vaginal branch* the remainder of the operation is practically bloodless." If the bladder is pushed off the anterior surface of the uterus to its interureteric fold the ureters are sufficiently far below the uterine arteries to be out of danger of

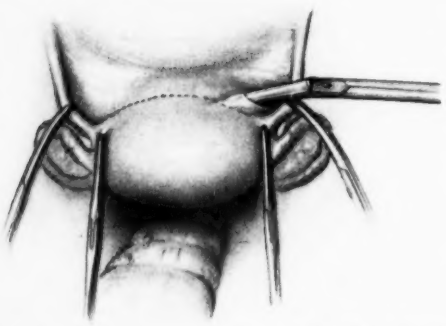


Fig. 7. The peritoneum in front of the right round ligament is nicked and blunt tipped scissors are introduced to push the bladder back.

injury. When the uterus is pulled upward and obliquely backward the uterine artery lies at nearly a right angle to, and now well above, the ureter. It is usually possible to see the course of the uterine artery after it is located at the lateral wall of the uterus, and to palpate it between thumb and forefinger before it is ligated just median to the ureter. A ligature at this point shuts off the arterial blood supply to uterus, cervix, and bladder, and a second ligature on the uterine artery as it enters the uterus shuts off the venous return. If the uterine artery is not visible a little gentle palpation or teasing of the tissues will find it, or if not successful the "trick," as Weibel terms it, will locate it, i. e. a deeper palpation with the fingers at the posterior part of the parametrium close to the uterus, as described years ago by Stimson. To quote Bonney again: "No forceps are needed in cutting through the cardinal ligament when this vessel is controlled." Considerable time is saved in operating by ligating the uterine artery at these two points. There is freedom from active bleeding and also from the continued venous oozing that is so troublesome. It eliminates too the placing of clamps deep in the pelvis in close proximity to the ureters at their entrance to the bladder.

4. Infection. The danger of infection from the vagina in performing total hysterectomy is, I believe, far less than the danger of infection in cutting across the cervix in the subtotal operation. There are no glands in the vault of the vagina, and therefore it is easier to disinfect this area than to render sterile a cervix that may harbor infection in the depths of its racemose glands, or to render sterile a sloughing growth in the cavity of the uterus that one may have to cut into in removing the body of the uterus. Read and Bell state that pulmonary embolism is the cause of one-third of the deaths following subtotal hysterectomy and only one-tenth of the deaths in total hysterectomy. Other writers place the mortality from embolism in subtotal hysterectomy as high as 50 per cent. A low grade infection is frequently the cause of thrombosis and embolism. The higher percentage of embolism in the subtotal operation may perhaps be due to infection from a diseased cervix or infection from the intra-uterine cavity. The thorough cleansing of the vault of the vagina

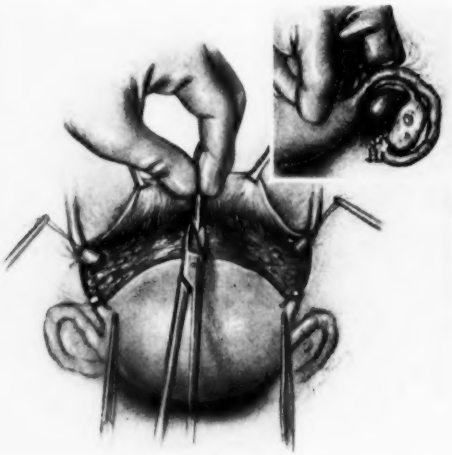


Fig. 8. Step. 5.

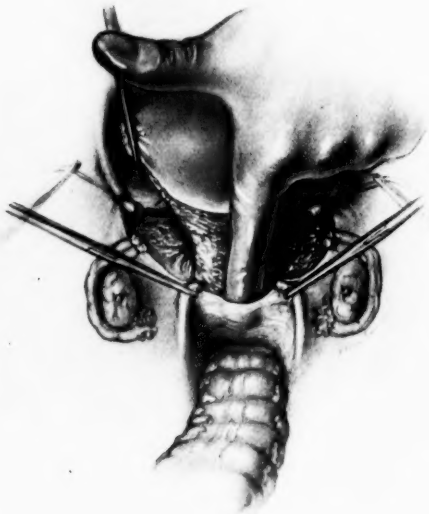


Fig. 10. Step 8.

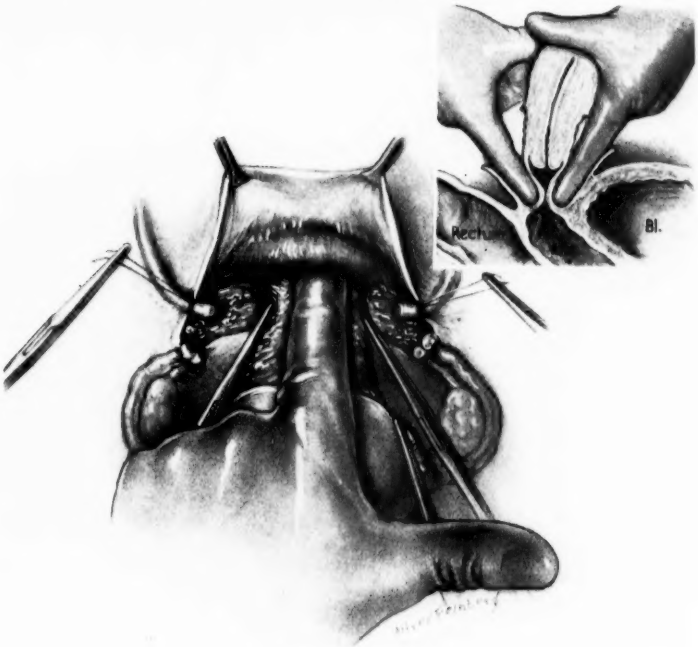
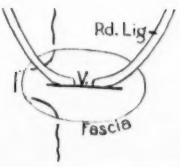


Fig. 11. Step 8.



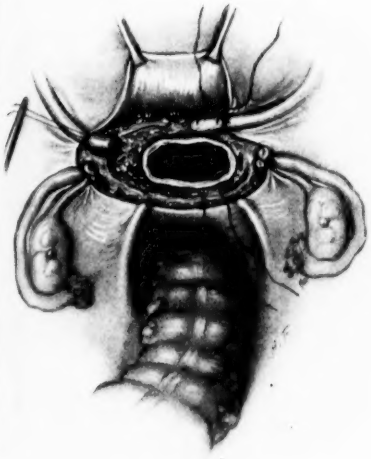


Fig. 12. Step 10.

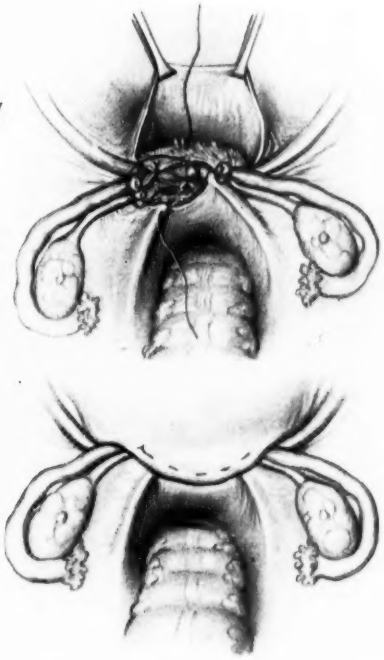


Fig. 13. Step 11.

is a most important part of the preliminary preparation of the patient and should be done personally by the surgeon, or at least supervised by him.

5. *Prolapse of vaginal tissues.* The shortening or prolapse of the vagina following total hysterectomy is due to the improper suturing or not suturing of the ligaments which were inserted into the uterus. When the uterus is cut away these ligaments should be fastened to one another and to the cut edges of the vagina. This prevents either the shortening or the prolapse of the vagina.

THE OPERATION OF TOTAL ABDOMINAL HYSTERECTOMY

The investigation of a patient's condition before operation should include the usual examination of heart, lungs, and also the throat for infection, the routine urinalysis and a phenolsulphonephthalein test of the kidneys. The blood count should be not less than 3,500,000 red cells and 70 per cent hæmoglobin, or the operation should be postponed until a blood transfusion can be given. The white cells should be between 5,000 and 10,000. The blood pressure of normal average for the age of the patient. All cervical erosions *must be healed before operation to prevent* infection. The patient should be in the hospital at least 24 hours before the operation to care for the intestinal tract and prevent the farewell lunches or dinner parties that are not infrequently the cause of much postoperative trouble. A sedative the night before operation and a preliminary hypodermic of morphine and atropine helps to quiet the nerves for the operation. If the patient is elderly or a poor risk I have been in the habit for several years of having a solution of gum acacia and glucose given intravenously during the operation as a supportive measure, and at the closure of the abdominal incision, to have given by rectum a quart and a half of saline solution, or what Dr. John Clark termed "an internal hot water bottle" to offset the loss of body fluids.

Step 1. When the patient is under the anæsthetic and in the lithotomy position, after the external genitalia have been cleansed and the bladder catheterized, the entire vagina should be thoroughly scrubbed out with sterile gauze sponges on long sponge forceps, tincture of green soap, and with plenty of hot water. Especial care should be given to cleaning around the cervix at the vault of the vagina. A douche of sterile hot water is then given, a long nozzle being used to reach to the vault of the vagina. A sterile weighted speculum, double tenaculum, Deaver vaginal retractor, cervical dilator, gloves, and long intrauterine syringe will be needed. An ounce of 3½ per cent tincture of iodine is injected into the uterine cavity and the iodine withdrawn into the syringe at the external os so as to avoid any washing out of cancer cells. The cervix and vault of the vagina are painted thoroughly with fresh iodine. The external os is sutured, and 3 yards of two-inch iodoform gauze is packed snugly into the vault and around the cervix. This packing helps lift the vault of the vagina when the cervix is cut out, and remains *in situ* 48 hours after the operation, as it keeps the tissues protected. There is no danger of sewing this gauze to the vaginal edge as there is so much elastic tissue in the vagina that it can be pulled up as soon as the cervix is cut out.

Step 2. After a median abdominal incision from the pubis to the umbilicus (or curved to right or left and then above if the abdominal wall is thick), the edges of the wound are protected with gauze, a self-retaining retractor is inserted, the patient is placed in the Trendelenburg position, and the intestines are packed back. (Three Bissell rubber pads or pockets filled with Turkish towelling are a decided help and do not mind paper the intestines as gauze packs do.)

Step 3. An artery clamp is placed on each round ligament close to the uterus and the tissue pulled taut. The peritoneum just in front (Fig. 7) of the right round ligament is nicked and blunt tipped scissors are introduced to push the bladder back. The vesical peritoneum is cut after the bladder is pushed away and several Allis clamps are placed on the cut edge. The round ligaments are then cut across and each tied, the suture being left 2 or 3 inches long with a clamp on each so the round ligament may be readily identified as they are to be used in the closure of the vagina.

Step 4. A sharp toothed Kocher forceps is clamped on each side of the uterus. (No sharp toothed or elevating forceps should be applied to the fundus as cancer may be present and unsuspected.) A small artery forceps is passed directly under each fallopian tube and the tissues pushed downward to allow the ureters to drop farther away. The tubes and ovaries are then removed or left as deemed best, the ovarian arteries being ligated at the infundibulopelvic ligament or at the uterus. I always leave normal ovaries if the pathologist reports no carcinoma in the fundus, and the tube with each ovary, if there is no appearance of infection in the tube, as there is less disturbance of the blood supply to an ovary if the corresponding tube is left. If the tubes are removed it is desirable to leave at least one ovary and if there is doubt as to its condition it may be placed just above the peritoneum when the abdominal wound

is closed. I have done this several times and so far I have never had to remove the ovary, nor do I know of any trouble ever resulting from having left it. Each ovary and tube is ligated separately to avoid mass ligatures. If the tube is to be left I wipe out the lumen of the tube with small cotton wound applicators dipped in carbolic acid and the last one is dipped in alcohol. All blood vessels are ligated by sewing in the ligature to prevent slipping. The entire field is cleared of clamps before proceeding to the next step, except the two Kochers on the uterus and the Allis clamps on the vesical peritoneum.

Step 5. The bladder is picked up with thumb and fingers and the vesical ligament is snipped with blunt tipped angle scissors, the incision being extended toward the uterus until the bladder is freed to the interureteric fold (Fig. 8). When this fold is reached we know that the bladder is free to the trigone and that the ureters have dropped down in the pelvis. One can usually see the ureters entering the bladder and palpate each between the fingers as a flat cord,—and it is almost always possible, except in the very fat patient, easily to trace the ureters through the parametrium.

Step 6. The uterine artery is located at its entrance into the lateral wall of the uterus. The uterus is pulled up and obliquely backward (Fig. 9) and with thumb and forefinger the uterine artery is palpated for approximately three-fourths of an inch from its insertion into the uterus. When certain that the blood vessels only are in the grasp of the fingers, while they are still held, a needle with suture is carried directly under the vessels and tied at this spot which is *median to the ureter*, at right angles to it and now well above the ureter. I suture the uterine artery again just as it enters the uterus, and then suture the uterine artery to the fascia.

Step 7. Each uterosacral ligament is cut just above its insertion into the fascia and a Kocher clamp is placed on each ligament. The fascia is cut at this level entirely around the cervix, going approximately one-fourth inch above the ligated uterine arteries. The parametrium is cut on one side to about one-half of its depth and then on the other side. The cutting is continued first on one side and then on the other until well down to the vagina. Usually when the parametrium is entirely cut through it is possible to pull the uterus up and out of the pelvis.

Step 8. To ascertain if the cervix is freed down to the vagina an incision is made between the uterosacral ligaments and the uterus is pushed forward to free it from the rectum (Fig. 10). In a similar way in front a finger is pushed down, pressing back on the uterus to free it from the bladder (Fig. 11). Test with a finger anteriorly and one posteriorly. If the lateral dissection has been continued downward sufficiently far the fingers will meet below the cervix.

Step 9. A bit of vaginal tissue is picked up between the uterosacral ligaments and nicked transversely. When the vagina is opened it is easy to push in a curved artery clamp, fasten it on the vaginal tissue, and cut the tissue close to the cervix. (I use a different clamp on the vaginal tissue from the one on the fascia simply to identify the edges of the vagina and work more quickly when suturing.) Repeat the procedure with clamp and cutting until near the bladder. Then the vaginal tissue is clamped and cut on the other side from posteriorly to the bladder. In this way the vaginal tissue near the bladder is left to the last when it will be easily seen and the bladder can be protected from injury. If one is in doubt how broad the cervix is and how far out to cut the vagina it is better to introduce a finger into the vagina rather than to put a hook into the cervix and draw it out as is often advised. When the cervix is entirely removed I apply iodine on a sponge to the vaginal edges and remove the excess iodine with a dry sponge. Up to the incision of the vagina the operative field will be almost bloodless if the ligatures have been properly applied. When the vagina is cut through there is some slight bleeding from the branches of the external vaginal arteries which supply the middle portion of the vagina and from the inferior hæmorrhoidal artery, but this is checked by the suturing of the vaginal edges.

Step 10. The edges of the cut vagina are closed with No. 2 plain catgut suture, beginning on the right posteriorly, passing the suture from without into the vagina and out at the extreme right end of the tissues and returning into the vagina and then out at the anterior edge of the vagina (Fig. 12). This secures the right side and I include at the same time a bite of the right round ligament and tie. A suture then from the posterior cut edge of the vagina to the anterior edge of the vagina closes the right half of the vaginal opening. In this suture I include again the round ligament, near its cut end. This firmly secures the vaginal tissues against prolapse. The steps are repeated on the left half of the vaginal opening.

Step 11. With the same type of suture the fascial edges are united, beginning posteriorly on the right by passing the suture from without through the fascial opening and out the extreme right end of the fascial tissue and back in this same tissue,

and then out through the anterior edge of the fascia exactly as in the closure of the vagina (Fig. 13). Next a suture is passed from without through the right uterosacral ligament (included in this suture is the cut ends of tube and ovary if they have been left) and through the anterior edge of the fascia and tie. This procedure is repeated on the left side of the fascial opening.

Step 12. There remains now only the peritoneum to be closed over the raw surfaces. This should be done carefully so that no raw edge is left for an adhesion to the intestine and possible ileus later. A Lembert suture introduced one-eighth inch from the cut edge of the vesical peritoneum will roll in this edge and leave a smooth surface when the peritoneum is sewed down to the raw tissues.

Closure of the abdomen is the final step unless the appendix or other work is to be included. Repair of the pelvic floor may be done best after the completion of the hysterectomy if the patient's condition seems then to warrant it.

This is the third year I have used this technique and so far there has been no hæmorrhage, no injury to bladder or ureters, and no prolapse of tissues. It requires only a few minutes longer to perform than the subtotal operation but is well worth the time. The postoperative convalescence is decidedly better than after the subtotal hysterectomy. The mortality up to now has been 1.8 per cent.

SUMMARY

1. To Dr. Lewis A. Stimson, a founder of and the first professor of surgery in Cornell University Medical College, belongs the credit for the following technique. (a) The locating of the uterine artery as it enters the uterus, and isolating of it before ligating when performing hysterectomy. (b) The covering of the pedicle or stump left in subtotal hysterectomy with a flap of peritoneum. (c) The recommendation of total hysterectomy which he performed in 1889.

2. This technique for total abdominal hysterectomy is based upon the anatomy of the endopelvic fascia and blood supply of the uterus, cervix, and vagina.

3. It lessens the danger of hæmorrhage, of injuries to bladder or ureters, and to infection from cutting across the cervix or into the uterine cavity.

4. It prevents shortening of the vagina or prolapse of the vaginal tissues after the operation.

5. Total abdominal hysterectomy avoids the danger of leaving a diseased cervix or an unsuspected carcinoma in the cervix.

6. The cervix left after a subtotal hysterectomy is devitalized by the cutting off of the blood supply, and like all devitalized tissue is liable to infection. Many patients who did not have a discharge before the operation suffer later from an annoying leucorrhœa.

7. A devitalized infected cervix is a menace to a patient as cancer of the cervix usually begins in a chronically diseased cervix. Many cases have been reported of cancer beginning in a cervix 1 year or more after a subtotal hysterectomy.

8. It is illogical to compare the mortality of the subtotal with the total hysterectomy as the conditions for which the two operations are done are very different. Most surgeons perform daily a supravaginal hysterectomy for fibromyomata of the uterus and do a total hysterectomy infrequently and usually only when it is known that cancer of the fundus is present.

9. For a fair comparison of the subtotal and total hysterectomies one must add to the primary mortality of the subtotal operation the subsequent morbidity or mortality of the removal of a diseased cervix, or radium treatment for a cervix that has developed cancer following hysterectomy.

10. Total abdominal hysterectomy has been performed by many surgeons with a mortality as low or lower than the subtotal hysterectomy and should be the operation of choice for benign conditions of the uterus necessitating hysterectomy.

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THE SURGERY OF GANGRENE OF THE EXTREMITIES

WITH A STUDY OF 171 CASES FROM THE RECORDS OF THE NEW ORLEANS CHARITY HOSPITAL[1]

J. ROSS VEAL, B.S., M.D., AND ELIZABETH M. McFETRIDGE, M.A., NEW ORLEANS, LOUISIANA

MERLE Scott, Mont Reid, and one or two other writers have repeatedly made the point that the field of arterial vascular disease has long been a neglected field and a badly handled field, and Reid, in a recent brilliant survey of the whole subject, goes even further. Students and surgeons, he says, are no longer satisfied with efforts to classify gangrene and to determine the optimum level of circulation; instead, they want to know the actual degree of circulatory embarrassment, and to evaluate the rôles of the vasomotor nerves, mechanical obstruction, infection, and the general condition of the patient. His attitude is both wise and hopeful, for gangrene, which is really an end-result, a terminal phenomenon, is very generally regarded as the initial manifestation of arterial obstruction, and its management is still characterized by an attitude of utter hopelessness that does not promise well for the results of treatment. That there is some justification for despair it must be admitted. The patient with arterial obstruction must be saved from his disease before gangrene sets in if he is to be saved from death or from mutilation, and that implies an entirely new conception of the condition on the part of the profession as well as of the laity. Reid and his associates at this time are voices crying in the wilderness, but they are also heralds of a new day.

On the other hand, however much one applauds their efforts and endeavors to emulate their methods, the fact remains that the treatment of vascular disease of the extremities among intelligent private patients and its treatment, or rather the treatment of its terminal phenomenon, gangrene, among ignorant public charges, are two very different matters. J. M. T. Finney, Sr., was quite correct when he remarked that anybody could amputate a leg but that it took a good surgeon to save one. He did not, however, go all the way; even the best of surgeons cannot save a leg that is beyond saving when it is first seen, or that can be saved only by an unjustifiable risk to life. In the public hospital the surgeon most of the time has just one course open to him, prompt amputation, for the reason that the patient most of the time presents himself too late to make conservatism safe or practical. If he resorts to amputation without delay he may—though he does not always—save the patient's life. If he practices watchful waiting, hoping to avoid the mutilation that is abhorrent to all his instincts and training, he frequently fails in his attempt at salvage, and he always introduces the risk of an increased morbidity and mortality. The surgeon who procrastinates, no matter how worthy his motives may be, adds to the death rate in gangrene just as inevitably as does the patient who delays presenting himself for treatment or who refuses surgery when it is offered to him.

But there is another problem besides the major one of whether or not to amputate. Where to amputate is a very serious consideration. Some surgeons, notably Eliason, still observe the Heidenhain law, that amputation is, as a rule, best done high, because at that level the presence of an adequate circulation is ensured. That there is ample ground for this position cannot be denied, at least in the absence of reliable tests to determine the lowest level of adequate circulation, but the consistent observance of this rule is open to objection on two points. The first, and more immediately important, is that in all amputations the mortality rises in direct proportion to the nearness of amputation to the trunk, although the risk of secondary gangrene is naturally very much less. The second is that it is seldom possible to fit an artificial limb satisfactorily above the knee, because, although a pressure-bearing stump can be obtained with little difficulty, leverage to manipulate the leg is lacking. That point we do not believe, as does Eliason, can be ignored, particularly in these days, when more than ever before economic usefulness must be taken into consideration.

During the 5 year period ending in December, 1933, there were 171 amputations of the extremities for primary vascular disease at the New Orleans Charity Hospital, of which 110 were for arteriosclerotic gangrene, 45 were for diabetic gangrene, 14 were for Buerger's disease, and 2, both in cardiacs, were for embolic gangrene involving the popliteal artery. Sixty-seven of these patients died, a mortality of 39.1 per cent, which, though it is about the mortality reported from other clinics handling the same type of case, is sufficiently high to demand earnest consideration, especially since preventive measures can elimi-

[1]From the Department of Surgery of the Louisiana State University School of Medicine.

nate much of it and can materially reduce the mutilating surgery which gives rise to it. We should be derelict, too, even though we have attempted no follow-up on these patients, if we did not emphasize a fact which Eliason called attention to in his most recent publication on this subject, that the immediate surgical and the deferred hospital death rates do not tell the whole story; to them must be added the proportion, the appalling proportion of patients who die within the year after their discharge and who cannot be ignored when the accounts are finally cast up.

The pathology of the various types of gangrene is so clearly understood today that only one or two practical considerations need be stressed here, the first of which is that whether the individual is suffering from arteriosclerosis *per se*, or from arteriosclerosis associated with diabetes, the fundamental pathology is precisely the same, its manifestations being different merely because in diabetes an additional metabolic factor is introduced. The essential sameness of these two types of gangrene was first pointed out by Buerger and by Eliason and Wright, and the correctness of their contention was immediately recognized. In both diseases the underlying pathological change is an obliterating endarteritis, frequently associated with calcium deposits, which affects all the vessels of the body from the thoracic aorta to the finest radicals. The narrowing of the lumen in the large vessels amounts, in the smaller vessels, to practical occlusion, and leads, in turn, to alterations in the nutrition of the tissues, especially of the kidneys, the brain, the myocardium, and the extremities, of which gangrene may be the end-result.

The fact that the diabetic exhibits his sclerosis at an earlier age by approximately 10 years than does the essential arteriosclerotic, a difference which was first emphasized by Eliason and Wright, we shall discuss in more detail later. Here we would merely point out that the metabolic perversion of the diabetic counterbalances, if we may so express it, the enfeebled heart and kidneys of the older arteriosclerotic and that their surgical risk is, from this standpoint, about the same. But the diabetic is peculiarly prone to infection, and when infection is once introduced insulin, which is his sheet anchor at other times, loses a large part of its effectiveness and his risk is many times increased. The reaction of the arteriosclerotic to infection is more sluggish, but it is by no means absent. It is present more often than is generally realized, but it is a deep seated infection, an underground process, if we may so express it, that spreads along tissue planes and that only rarely manifests itself by the line of demarcation for which, traditionally, the surgeon waits and because of the delay in the formation of which so many lives have been sacrificed. In both arteriosclerotic and diabetic gangrene, infection is always actually or potentially present. The arteriosclerotic may react to it more sluggishly, his gangrene may begin as the dry type, in contradistinction to the moist and always potentially infected gangrene of the diabetic, but in both diseases a vicious circle is likely to be established in which infection increases gangrene and gangrene increases infection.

The constitutional reaction in the diabetic is, case for case, more marked than in the arteriosclerotic. The temperature rises, the pulse is accelerated, and the increase in the blood sugar, the development of glycosuria, and the tendency to acidosis which result from the local condition are exceedingly difficult to control by routine or even extraordinary measures. In the arteriosclerotic, the rise in temperature and in pulse rate is rarely marked in the early stages, and is, at any time, a poor index of the amount of infection and toxæmia present, as the surgeon who so regards it frequently finds to his cost. The added decade of life, with all that it implies in the way of decreased function of the vital organs, introduces an immediate added risk, and a clinical appreciation of the patient's general well-being, or, more correctly, his lack of well-being, is a better guide to his condition than are the usual signs. In these patients toxæmia is manifested by an indifference to food, a markedly reduced fluid intake with a corresponding reduction in the fluid output, a listlessness and drowsiness that at times approach actual stupor, and an involvement of the cerebral vessels manifested in many cases by a mild senile dementia. This picture we have been particularly impressed with in the patients in our colored male wards, and the improvement in our results since we have realized its significance and have sought to correct it, especially by forced fluids before and after operation, has been most gratifying.

Buerger's disease, or thromboangiitis obliterans, which is a presenile inflammatory disease of the vascular system characterized by gangrene of the fingers and toes rather than by massive gangrene, exhibits, as a rule, premonitory signs and symptoms which cause the patient such extreme discomfort that he usually presents himself for treatment long before frank gangrene impends. The possibility of gangrene in the diabetic is always present in the mind of a careful physician, provided that the diabetic has presented himself

for treatment, which is not always the situation. Eliason reports that more than half the patients with diabetic gangrene in his series did not know they had diabetes, and we found almost the same proportion in our own cases. The danger to the diabetic of minor trauma, such as arises from paring calluses, cutting corns, wearing tight shoes or shoes with rough linings, or even stubbing the toe, has been repeatedly emphasized, but we do not believe that the almost equal danger of such slight insults to the arteriosclerotic has been sufficiently stressed. Moreover, it has only recently been realized that patients with arteriosclerosis frequently exhibit signs of their disease which, if properly interpreted, can serve to prevent graver consequences. Reid, more than any other writer, has stressed the fact that intermittent claudication, pain in the feet and legs which is aggravated by walking and is worse at night, numbness and coldness of the extremities, swelling, discoloration, and similar signs and symptoms all demand prompt attention and treatment because they are all warnings of impending trouble.

We were particularly interested in this phase of the cases we studied. In 139 of the 171 cases it was possible to determine from the history the precipitating cause of the gangrene, which proved to be trauma in 43 (of 91) cases of arteriosclerotic gangrene, 28 (of 41) cases of diabetic gangrene, and 2 (of 7) cases of Buerger's disease. That is, trauma of some sort precipitated the gangrene in 73 of 139 cases in which the history was adequate. Furthermore, in more than half of the cases in the arteriosclerotic group the history showed that arterial disease had been present and could have been treated long before the gangrene made its appearance, for the signs and symptoms listed by Reid were noted again and again. Patients cannot be treated, of course, who do not present themselves for treatment, but at that, one looks with regret on the lost opportunities in this particular group of cases, on the fatalities that could have been averted, and on the radical surgery that had to be done because those opportunities were lost.

Lemann, writing in 1927 on gangrene in the South, called attention to poor pedal hygiene as a very important cause, particularly among negroes, and we desire to stress that point again. Indeed, we could scarcely avoid stressing it, for 107 of these 171 cases, 62.5 per cent, occurred in negroes, as did 42 of the 67 deaths, 62.6 per cent. All the points that Lemann made 7 years ago still hold. Negroes wear cheap and badly made shoes, which are full of unevenness if not of actual holes, their socks, if they wear socks, are in like condition, their feet are badly kept and seldom washed, and, partly because of the color of their skin, partly because of their habitual uncleanliness, minor abrasions and injuries, and even initial gangrene, are frequently overlooked. The negro's habit of smearing himself with salve for all local conditions is often at least a partial explanation of the infection which usually accompanies his gangrene, while his habit of delaying treatment for even serious disease is too well known to need extended comment. It does seem obvious, however, from what we have just said, that it is quite as important to advise the arteriosclerotic in the care of his feet as it is to advise the diabetic.

The difference between the white and negro incidence and mortality is so striking, as we have just noted, that it is unfortunate that no comparative figures are available. In our own series no cases of Buerger's disease, as would be expected, occurred in the negro, but 82 of the 110 cases of arteriosclerotic gangrene were in that race, as were 25 of the 45 cases of diabetic gangrene. In Eliason's last report of 175 cases of diabetic gangrene the negro to white ratio was 1:4, but a comparison with our own statistics means nothing in view of the different racial proportions in the two cities, not to mention the fact that the negro of the South and the negro of the North are in all respects different. Lemann, writing in 1927, pointed out that in the preceding 6 year period 1 of every 5 negro diabetics applying for admission to the New Orleans Charity Hospital exhibited gangrene, the negro incidence of that complication being 21 per cent against the white incidence of 14 per cent. In our own figures, which cover the period from 1929 through 1933, we found the disproportion still existing, though it was less striking. Forty of the 310 cases of diabetes in the negro were associated with gangrene, 13 per cent, against 44 of the 582 cases of the disease in the white, 7.56 per cent, the reduction undoubtedly being due to the wider and more intelligent use of insulin in these later years. The mildness of our climate, too, may play some part, for our general incidence for both races of 9.4 per cent is decidedly less than the 13 per cent which Eliason reports in his cases.

The incidence of syphilitic disease in the Southern negro is notorious, and syphilitic patients, it is generally admitted, are prone to develop arterial disease earlier than their uninfected brothers. Joslin many years ago made the point that diabetic patients with gangrene show a positive Wassermann seven times as frequently as do diabetic patients without gangrene; in his whole

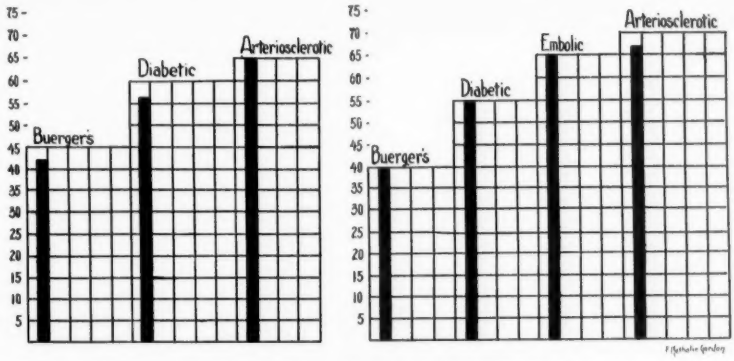

Fig. 1. Left, average age of onset in various types of gangrene. Right, average age of death in various types of gangrene.

series only 2.8 per cent of all diabetics exhibited gangrene against 10 per cent of syphilitic diabetics, but these figures Lemann was unable to corroborate for the South. In our own series, however, 16 patients in the senile arteriosclerotic group of 110 cases, 13.6 per cent, had positive Wassermann reactions, and 12 of them were negroes. Two of the 45 diabetics, both negroes, also had positive tests, and we have no doubt that the number in both groups would have been materially increased had the past history of syphilitic infection been considered as well as the present laboratory manifestation.

Eliason's and Wright's statement that diabetic gangrene ensues approximately 10 years earlier than the arteriosclerotic variety we have found to be true, as Figure 1 shows. The average age in the arteriosclerotic group was 64.2 years, 66.7 years for the white against 63.7 years for the colored, while in the diabetic group the average age was 56 years, 64.5 years for the white against 51.9 years for the colored, the discrepancy between the white and the colored age of onset being particularly striking in the diabetic group. In this same connection, it might be mentioned that the arteriosclerotics with positive Wassermann reactions exhibited their gangrene at an average age of 61.3 years and the diabetics at an average age of 42 years, which is well below the average age for each group. The single patient with Buerger's disease who had a positive Wassermann reaction was only 28, although the average age for the group was 42.5 years.

Since the risk of surgery for gangrene increases with age, it is not surprising to find (Fig. 1) that the average age of death in the arteriosclerotic group was 67.8 years, more than 3 years more than the average for the whole group. The age limits of the arteriosclerotic group ranged from 43 to 91 years, as contrasted with 37 to 84 years in Eliason's group, and from 32 to 74 years in the diabetic group, as contrasted with 34 to 79 years in Eliason's group.

The arteriosclerotic variety of gangrene is overwhelmingly more frequent in the male, 90 against 20 cases, while the diabetic variety is more frequent in the female, 28 against 17 cases. All the cases of Buerger's disease occurred in white males. It is rather interesting that the average age of the females with arteriosclerotic gangrene was 69.2 years, while the male age was considerably lower, 63.4 years, and that in the diabetic group the average age of the female was 55 years against a male age of 51.3 years. The latter discrepancy is worth noting in view of Eliason's and Wright's conclusion that the female with diabetic gangrene runs more risk at an earlier age than does the male with the same condition.

Table I, which shows the mortality in relation to the underlying disease, is self-explanatory. From Figure 2, which shows the mortality and the development of secondary gangrene in relation to the site of amputation, certain conclusions may be drawn. In arteriosclerotic gangrene, if one is sure that the vascular occlusion is confined to the digital branches, conservatism can be safely

TABLE I.—MORTALITY IN RELATION TO DISEASE

Disease	Cases	Deaths	Mortality rate
Arteriosclerotic	110	43	39.0
Diabetic	45	19	42.2
Buerger's	14	3	21.4
Embolic	2	2	100.0
Total	171	67	39.1

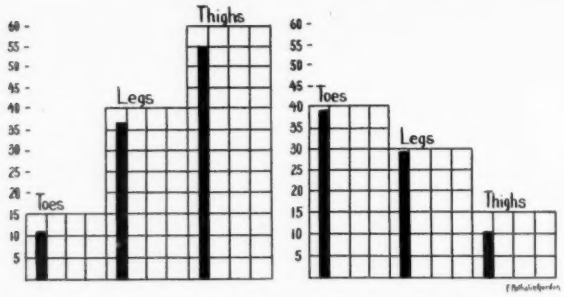

Fig. 2. Left, mortality rate of amputation at various levels. Right, Incidence of recurrent gangrene at various levels of amputation.

practiced, for the chances are that it will be successful, and the risk of reamputation, though naturally greater, is still not excessive. In the diabetic, on the other hand, in whom the initial disease is more serious and in whom the tendency of the wound to heal is militated against by his constitutional disease, the situation is much more serious; it apparently cannot be taken for granted that the gangrenous process is localized, and reamputation carries with it a very serious risk. Finally, the point we made originally is corroborated by these figures, that the higher the amputation is done, the less is the risk of recurrent gangrene, but the greater is the mortality. In Eliason's series reamputation in arteriosclerotic gangrene carried a mortality of 10 per cent against an original mortality of 34.2 per cent, whereas in the diabetic group it carried a mortality of 50 per cent against a mortality of 35.1 per cent for the first operation.

The 14 cases of Buerger's disease deserve some special comment. In the 2 cases in which fingers or toes were amputated at intervals, there was no mortality. In the 7 cases in which amputation was done on the leg there were 3 recurrences, necessitating 2 reamputations, and there was 1 death, from a streptococcic infection of the stump. In the 5 cases in which amputation was done at the thigh, there was 1 instance of recurrent gangrene, and 2 deaths, 1 from lobar pneumonia in 4 days, and another 37 days after operation from an infection of the stump associated with pulmonary embolism and septic infarcts of the lungs, liver, and kidneys. The high percentage of recurrences seems to prove the point made by a number of writers that amputation in this disease should be deferred until the limits of the process are clear.

Studying the mortality from the point of view of the time at which it occurred, we find that 9 of the 43 deaths in the arteriosclerotic group occurred within 24 hours, from shock, usually associated with toxæmia, in 8 cases, and from cardiac failure in 1. Eight of the 19 diabetic deaths occurred within 24 hours, from shock in 4 cases, the diabetes being under perfect control, and from diabetic coma in the other 4, associated, in 1 case, with a pre-existing gas bacillus infection. The immediate surgical mortality, therefore, in these two groups is 17, 9.9 per cent, as compared with 3.6 per cent in Eliason's and Wright's 1926 series, and 3.5 per cent in Eliason's 1933 series, which includes only diabetics, this disproportion being rather interesting in view of the fact that the final mortality rates are not very different, 39.1 per cent for the Charity Hospital cases against 33.3 per cent for Eliason's arteriosclerotic cases, and 43.6 per cent for his diabetic cases.

Of the 19 arteriosclerotic deaths which occurred from 2 to 10 days after operation, 10 were due to pneumonia, 4 to uræmia, 2 to cardiac failure, 2 to recurrent gangrene and toxæmia, and 1 to gas bacillus infection. Of the 7 diabetic deaths which occurred within the same period, 3 were due to diabetic coma, 3 to recurrent gangrene associated in 2 cases with septicæmia, and in 1 with pulmonary abscesses and otitis media, and 1 to pneumonia.

Of the 15 arteriosclerotic deaths which occurred after 10 days, 9 were due to pneumonia, 2 to cardiac failure, 2 to renal complications, and 2 to recurrent gangrene and toxæmia. Of the 4 diabetic deaths which occurred in this period 2 were due to diabetic coma and 1 each to recurrent gangrene and toxæmia and to myocardial failure and cerebral embolism.

To summarize, and as Figure 3 shows, in the arteriosclerotic group, shock and pneumonia were responsible for the greatest number of deaths, while in the diabetic group diabetic coma was

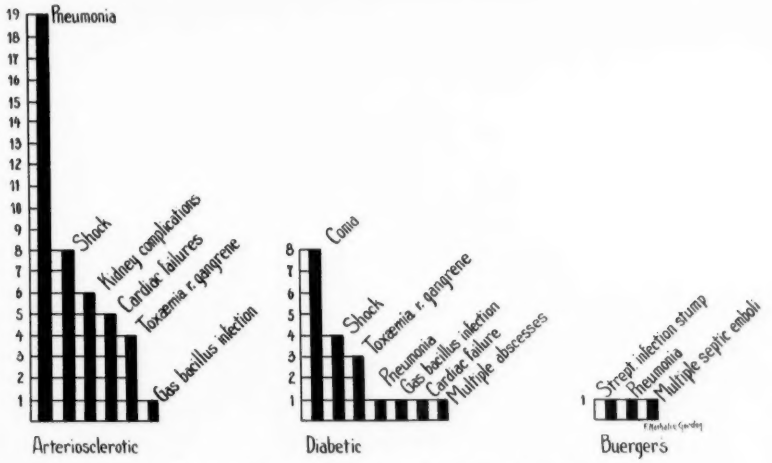

Fig. 3. Causes of death in various types of gangrene.

responsible for almost 50 per cent. The contrast with the arteriosclerotic group is illuminating but rather shocking in these days when diabetic coma is supposedly a condition within medical control. Undoubtedly the explanation is the tendency of these patients to delay entrance to the hospital, or to refuse surgery when it is offered to them. The last 2 diabetics who died on our own service died because they themselves refused operation until it was too late, not realizing that in this disease the mere existence of gangrene and infection constitutes a threat to life and that the risk increases with every day that operation is postponed.

Only 2 cases in this whole series developed gas bacillus infection, which was surprising to us in view of the 14 cases reported by Eliason, 10 of them postoperative, in his 175 cases. There is undoubtedly in this disease a sort of seasonal incidence; our statistics end with December 31, 1933, and during January and February of 1934 we had on our own service 3 patients with gas bacillus infection following amputation, 2 of whom died and the inclusion of whom would have materially altered our percentage. How or why these patients developed the infection we do not know, though its occurrence in the cold months, when blankets are in use, naturally makes us think of Gage's brilliant work on the introduction of the infection from the contact of an open wound with woolen goods, even after it has gone through all the processes of manufacture. That more of our colored patients do not develop this particular contamination is rather surprising, for the ward is overcrowded, they have the most elementary ideas of hygiene, and the soil seems ripe for it.

Certain explanations for our very high death rate immediately present themselves. Age is a factor which cannot be controlled and patients advanced in years, with associated degenerative diseases of the heart and kidneys, are bound to exhibit a higher mortality than younger subjects. Furthermore, they endure rest in bed badly, both before operation and after, and are particularly prone to develop a hypostatic pneumonia which is likely to carry them off. This was recently proved by one of our own colored male patients. He entered the hospital in a deplorable state, which is not unusual, he refused operation for more than a week, and he developed a postoperative gas bacillus infection which, due to the carelessness of an interne, was not reported to the staff man for more than 8 hours. For several days following the second amputation he was seriously ill, and he weathered the postoperative storm only to lose his life 10 days later from a hypostatic pneumonia.

Our figures show that the duration of the gangrene, provided toxæmia and infection are not associated factors, has surprisingly little effect on the mortality rate, but that its extent (Fig. 4) is the most important single consideration in determining the outcome. For that extent, as we have said several times, the surgeon is sometimes responsible but the patient more often is, partly because he delays seeking medical advice, partly because he refuses to follow the advice that is

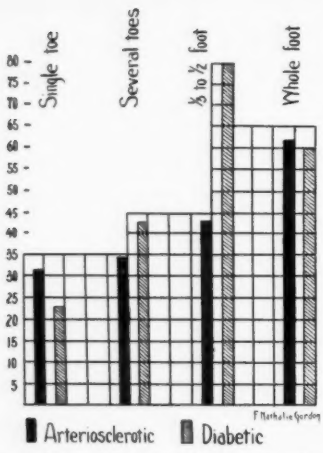

Fig. 4. Relation of extent of gangrene and mortality rate.

given him. Once gangrene is established beyond hope of recovery, the single most important factor in reducing the surgical mortality is the promptness with which operation is done. Two things cannot be too often emphasized, that a diabetic may lose his life because the optimum time for operation is allowed to pass by, and that neither diabetics nor aged arteriosclerotics tolerate toxæmia well. The degree of the constitutional reaction seems almost as important as the extent of the gangrene. Eighty-two per cent of the patients who died in this series had a febrile reaction above 100 degrees F. before operation, while only 25 per cent who lived exhibited it; the longer the reaction endured, the higher the mortality.

Careful pre-operative preparation is essential. The preparation of diabetic patients is standardized and should be in the hands of a competent internist, who works in full accord with the surgeon. Medical matters are the business of the internist, but the surgeon must remember that when the diabetic is sick of other diseases, particularly diseases in which infection is a factor, the usual reliance cannot be placed on insulin and his work must be done as expeditiously as possible. In the arteriosclerotic group, as we have already mentioned, we have noted an encouraging fall in the mortality since we have begun to study before operation the kidney function and general condition and to force fluids before and after operation. We are careful also to move these patients about in bed, and to get them up as promptly as possible, to guard both against pneumonia and against decubitus ulcers.

We do not believe that any death in this series could be attributed to the anæsthesia used, although there seems no question that spinal analgesia, or nitrous oxide or ethylene anæsthesia, is the agent of choice. Ninety-nine cases were done under spinal analgesia, 37 under ethylene, the remainder under local ether or nitrous oxide.

The type of amputation seems to have considerable bearing upon the recurrence of gangrene and the development of sloughs, which is to be expected, for many of these patients have an actual or a latent infection when the operation is done, and postoperative infection is likely in any tissues in which the circulation is impaired. In our own opinion, the simple circular type of amputation is wisest for all amputations except those on the fingers and toes, and a careful study of these records convinces us of the correctness of our contention. This is the old *en saucisson* method of the French writers, which was described several years ago by Urban Maes in a paper on surgery in diabetics. The chief point of the operation is that the lines of section are made at right angles to the long axis of the limb, which means, since the blood vessels of the extremities in passing from tissue plane to tissue plane do so more or less at right angles, that no tissue is left without its blood supply. The procedure is quite simple technically. The skin is incised, then allowed to retract. The muscles are sectioned at the level of the retracted skin, then allowed to retract. Finally, the bone is sectioned at the level of the retracted muscle. To express it differently, in each plane the section is made at a higher level, so that the final effect is that of an inverted cone. With this technique, with individual rather than *en masse* ligature, and with loose closure by three or four sutures over the face of the stump, it is rarely necessary to employ drainage.

SUMMARY AND CONCLUSIONS

1. Gangrene of the extremities in private and public practice introduces very different problems. A new conception of arterial vascular disease is essential on the part of both public and profession, but the mortality is not likely to be reduced, nor is the incidence of mutilating surgery, until patients present themselves earlier for treatment and until physicians recognize the significance of premonitory symptoms.

2. One hundred seventy-one cases of various types of gangrene have been studied in detail, with particular relation to the pathology exhibited, the predisposing causes, the racial incidence, the mortality of amputation, and the recurrence of gangrene after it.

3. The high death rate is attributed to delay in seeking treatment and in accepting surgery, and to the age and the concomitant cardiac and renal diseases of arteriosclerotic patients and the metabolic disease of diabetic patients, the extent of the gangrene being the single most important factor. Anæsthesia, other than ether, seems to play no part in the mortality, but the type of operation does, and the simple circular amputation is the one that is recommended.

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A POSITIVE TREATMENT FOR FRACTURES OF THE SHAFT OF THE FEMUR

A Preliminary Report Emphasizing Ambulatory Treatment

R. A. GRISWOLD, M.D., F.A.C.S., Louisville, Kentucky
From the Department of Surgery, Louisville City Hospital and the University of Louisville School of Medicine

FRACTURE of the shaft of the femur presents a problem in mechanics which is often difficult of solution. The almost universal method of traction and suspension is far from ideal. There is no positive control of either fragment and accurate reduction is difficult and sometimes impossible. Frequent readjustment of the apparatus is essential to maintain position and requires almost hourly care. Long hospitalization is the rule and is a distinct economic drawback. Reduction and fixation by plaster cast is satisfactory in cases in which the fragments can be locked. Often, however, slipping and displacement occur within the cast. These considerations have led many surgeons to advise open reduction with internal fixation as a routine measure. Experience with a new method of treatment of fracture of the tibia and fibula has led to the development of a somewhat similar procedure for the femur. For fractures of the tibia and fibula, countertraction is applied by a steel pin through the proximal fragment, and traction, rotation, and angulation by a pin through the distal fragment or os calcis. Incorporation of these pins in a plaster cast effectually prevents subsequent displacement. In the application of this principle to the femur, traction and countertraction have been applied at the ankle and perineum. Pins through the proximal and distal fragments have been used only for manipulation and fixation.

Deformities in fracture of the femur are due to the fracturing force and to muscle pull. The latter replaces the former when the length of the limb is restored by traction and is, therefore, the more important (Fig. 1). The deformities are shortening, rotation, angulation, and transverse displacement. They may be influenced by the direction of the fracture plane. Shortening is produced by the entire group of longitudinal thigh muscles and presents few difficulties if efficient extension is available. The proximal fragment is usually rotated outward, especially in high fractures, by the action of the external rotators of the hip. The distal fragment may be rotated in either direction by the weight of the foot but is usually everted. After the normal length of the limb has been restored and rotation corrected, displacement may be viewed from anterior and lateral aspects. From the anterior aspect it is due to the action of the abductors of the hip and the adductors attached to the shaft. If the fracture is high, the proximal fragment is abducted and the distal fragment adducted, thus producing outward bowing. If low, the proximal fragment is often adducted in relation to the distal fragment which is held in line by traction. From the lateral aspect the deformity is due to anterior flexion of the proximal fragment by the iliopsoas and posterior flexion of the distal fragment by the gastrocnemius group. Flexion of the proximal fragment is most marked in high fractures and of the distal fragment in low fractures.

The first principle of the usual non-operative treatment is to restore the length of the limb by extension. This corrects transverse displacement by the squeezing effect of the taut musculofascial tube in which the bone lies. The second principle is to bring the distal fragment into line with the proximal, leaving them both in their positions of muscle equilibrium. This is accomplished from the anterior aspect by abducting or adducting the distal fragment. From the lateral aspect the lower fragment is brought into line by flexing the knee to relax the gastrocnemius and lifting the fragment anteriorly to appose it to the flexed proximal end. Rotation is easily controlled by various means. Maintenance of the fragments in this position is then attempted by continuous traction or a plaster cast.

The method here presented gives accurate mechanical control of the proximal as well as the distal fragments. Firm, uninterrupted fixation is provided until healing occurs. The position is that of normal weight bearing rather than that of muscle equilibrium. Immediate ambulatory treatment is possible. The latter is important aside from the economic advantage of short hospitalization. Weight bearing encourages healing and prevents to a large degree the atrophy associated with immobilization. The natural physiotherapy obtained by the use of the extremity is superior to any form of artificial physiotherapy in maintaining the nutrition and tone of the tissues. When a walking cast is removed the strength of

the extremity and the range of motion at all joints is often surprising. It is not unusual to have immediate complete range of motion at the hip and ankle and 45 degrees of painless active motion at the knee. Measurement of the circumferences of the thighs and calves show little or no atrophy. This is superior to the state after removal of the usual padded, non-ambulatory cast, and at least equal to that following traction-suspension methods.

PROCEDURE

1. Under spinal or local anæsthesia the patient is placed on the fracture table and extension applied until the length of the limb is restored. This is obtained by Collin's hitches about the ankles and countertraction against the ischium of the *well side.*

2. After preparation of the skin, a steel pin, ⅛ inch in diameter and 6 to 8 inches long, is inserted transversely through the distal fragment just above the adductor tubercle. A second pin, 5/32 inch in diameter and 8 to 10 inches long, is inserted in the anteroposterior direction at or slightly below the level of the lesser trochanter. If placed above this point, the soft cancellous bone will not stand the strain imposed upon it. This pin is well lateral to the femoral vessels and sciatic nerve, the only important structures in this region. Accurate placement is best aided by the fluoroscope. These pins are inserted directly through the skin. Incisions are unnecessary and invite infection. Restoration of the length of the limb before insertion of the pins is important to prevent skin tension. Small alcohol or collodion dressings may be applied about the pins.

3. The distal pin is slung from the suspension bar of the table by a suitable caliper and lifted to correct flexion of the distal fragment. Flexion of the proximal fragment is corrected by a clockwise motion of the upper pin which is held manually in a caliper (Fig. 1a). From the anterior aspect the deformity is corrected by abduction or adduction of the distal fragment combined with medial or lateral traction on the proximal pin (Fig. 1b). Rotation is corrected by appropriate motion of one or both pins.

4. The position is checked by fluoroscopy or roentgenograms in two planes and any necessary residual correction carried out. It is occasionally advisable to overextend the extremity during manipulation so that there is a space between the fragments. This facilitates reduction in irregular fractures, but as soon as proper position is obtained, traction should be decreased so that the fragments firmly abut each other.

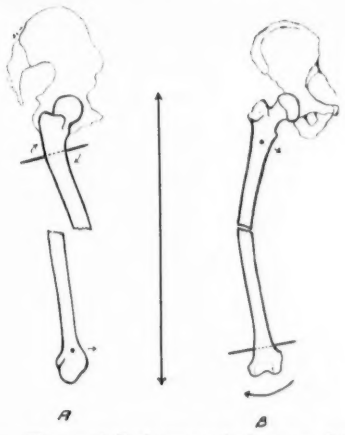

Fig. 1. The usual displacement in fracture of the shaft of the femur after length of the thigh has been restored viewed from A, the lateral aspect and B, the anterior aspect. The sites and positions of the pins are indicated. Arrows show the directions in which force is applied to the pins for reduction.

5. A non-padded walking spica is applied following the method outlined by Boehler and Schnek. The anterior superior spines and sacrum are protected by thin padding which is covered by a circular layer of flannelette extending from the xiphoid to the symphysis. A previously prepared roll of padding is applied to the tuberosity of the ischium for counterpressure. This is made by tightly rolling an 8 by 5 inch oblong of silence cloth over the center of a 3 foot length of bandage and covering it with adhesive tape. The pad is about 8 inches long and the thickness and firmness of a Thomas splint roll. The long ends of the bandage are pulled snugly upward over the flannelette so that the pad impinges firmly against the tuberosity. Circular plaster bandages are applied to the trunk and pelvis and molded about the crest and spines of the ilium. Anterior and posterior splints are applied to the skin from the Collin's hitch well up over the pelvic portion of the cast and covered with circular plaster bandages closely incorporating both pins. On the perineal side of the thigh, plaster is modeled over the ischial roll to form a shelf-like structure which holds the roll in place and transfers some of the pressure of weight bearing to the ischium after the manner of a Thomas walking caliper. The remainder of the weight is taken up by the pins. The junction of the thigh and pelvic portions of the cast is strengthened by additional splints and bandages. When this plaster has set the Collin's hitch is removed and the cast extended.

An anterior plaster splint is applied as far as the base of the toes and a posterior one to a point ½ inch beyond the tips of the toes. These are covered with circular plaster making firm union with the rest of the cast. A Boehler walking iron is incorporated as soon as the plaster has hardened. Trimming of the cast is carried out to uncover the upper abdomen and allow free motion of the uninjured thigh. Accurate and proper molding of this type of cast is essential, especially about the pelvis, knee, and ankle. One should have considerable experience with the smaller, non-padded casts before attempting a spica. The cast should be as light as is consistent with strength. Properly constructed for an adult of average size it weighs 12 to 14 pounds, and is considerably stronger than an equally heavy padded cast on account of the more evenly distributed stresses (Fig. 2, 10, 13).

6. Weight bearing is allowed as soon as the plaster is sufficiently hard and roentgenograms have checked position in the cast. In healthy young adults and children this is usually about the third day. Two crutches are used at first, later to be exchanged for canes as the patient gains confidence. Children not infrequently require no external support after a week or two. Weight bearing should be painless after the first few days. If it is not there may be some defect in the cast or reduction. The most probable cause of pain is insufficiently firm fixation of the pins in the cast allowing motion. The cast remains in place throughout the period of healing and may take the place of a convalescent walking splint. The pins may be removed at the end of about 8 weeks.

7. Feeble elderly individuals may be too weak to carry a cast and so not able to avail themselves of the advantages of ambulatory treatment. In this case the pelvic portion of the cast may be omitted or removed leaving the ischial pad and maintaining firm fixation of the proximal pin. It is possible that pivoting of the proximal fragment on the pin might occur with resulting outward bowing. This has not occurred in two cases so treated, but I feel that omission of the pelvic cast is advisable only if the patient cannot walk, and when it is essential to allow him to sit up in bed or a wheel chair.

APPLICATION OF METHOD

Putti has utilized a method for operative lengthening of the femur in which he places proximal and distal pins for traction and counter-traction. It should be noted that in the technique for fractures described in this paper no traction is applied to the pins so that they are left perfectly free for manipulation. The evolution of this method of treatment was necessarily gradual. The reduction of suitable fractures by manipulation and fixation in a spica cast was the natural starting point. The first additional step was to correct the posterior angulation of the distal fragment in fractures of the lower third of the femur by a steel pin as described by Anderson. In cases so treated bowing was found to occur with weight bearing. This was due to settling of the thigh into the cast despite the ischial pad (Case 1). The next development was skeletal control of the proximal fragment making the method applicable to all fractures of the shaft and effectually preventing displacement or bowing. "Mechanical hands" which may be firmly attached to "arms" from the table have been designed to manipulate and hold the pins until the plaster has set. These will eliminate the cumbersome method of hanging the distal pin from the suspension bar of the table by a rope and manually controlling the proximal pin. Some surgeons are not familiar with or are not convinced of the advantages of the non-padded cast and ambulatory treatment. For their benefit the pins may be used with the padded type of cast, adding the advantages of accurate reduction and firm fixation to their previous techniques.

This method has been applied to 10 cases. Five of these were not made ambulatory because of psychosis (2 cases), other fractures (2 cases), and transfer to another hospital shortly after reduction (1 case). The 5 other patients first walked with weight bearing on the second to the fifth day except one patient with a compound (gunshot wound) fracture who walked on the eighth day following fracture and the first day after application of plaster. Healing in good position with excellent functional result is known to have occurred in all these cases except the one transferred elsewhere. Hospitalization has been reduced to a minimum, averaging about 12 days for patients who did not have co-existing disease or injury for which hospitalization was required. The following cases have been selected to illustrate (1) anterior bowing due to weight bearing when the proximal pin was not used (Case 1), (2) use of the procedure when the patient cannot be made ambulatory (Case 2), and (3) typical ambulatory treatment (Cases 3 and 4).

CASE REPORTS

CASE 1. A. P., aged 18 years, Hospital No. A 7,496, was admitted with an irregular transverse fracture at the junction of the lower and middle thirds of the left femur with acute posterior angulation of the distal fragment and no

Fig. 2.

Fig. 3.

Fig. 4.

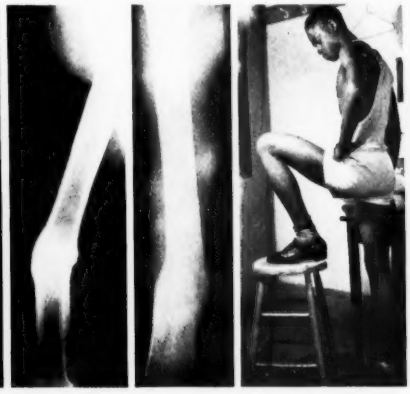

Fig. 5.

Fig. 6.

Fig. 7.

Fig. 8.

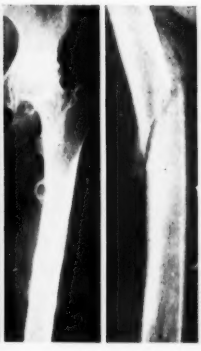

Fig. 9.

Fig. 10.

Fig. 2. Case 1. A plaster spica 1 month after ambulatory treatment was begun. The distal pin is shown covered with corks and plaster. Note the counterpressure pad at the groin.

Fig. 3. Case 1. Fracture of lower third of femur with typical displacement. No proximal pin was used in this case.

Fig. 4. Case 1. The fracture has healed, but bowing occurred as the result of settling of the upper fragment in the cast.

Fig. 5. Case 1. Function at the end of 5 months. Anterior bowing is clearly shown.

Fig. 6. Case 2. Fracture of middle third of femur and multiple fractures of other bones prevented ambulatory treatment. Treated with 2 pins.

Fig. 7. Case 2. Position and healing at the end of 4 months. The site of the distal pin is clearly visible.

Fig. 8. Case 3. An oblique fracture of the upper third of the femur with minimal displacement. Co-existing arthritis of the hip is also shown in Figure 9. Ambulatory treatment with 2 pins.

Fig. 9. Case 3. Position and healing at the end of 3 months.

Fig. 10. Case 3. Patient walking on crutches 1 week after injury.

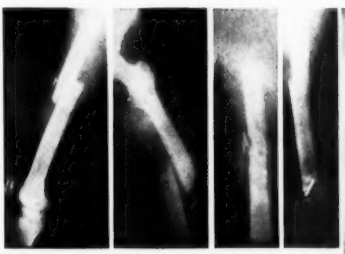

Fig. 11.

Fig. 12.

Fig. 13.

Fig. 11. Case 4. Fracture of middle third of femur. Ambulatory treatment with 2 pins.

Fig. 12. Case 4. Position in cast during ambulatory treatment.

Fig. 13. Case 4. Note molding of cast about the ilium and the plaster shelf covering the counterpressure pad. The covered end of the proximal pin is shown opposite the lesser trochanter. The distal pin is clearly visible.

apposition (Fig. 3). Under spinal anæsthesia the length of the limb was restored by traction on the Hawley table and the displacement corrected by manipulating a steel pin which was passed through the distal fragment. A Boehler spica was applied incorporating the pin. He was allowed up on crutches on the fourth day and discharged on the eleventh day. Roentgenograms at this time showed acceptable alinement and position except for some anterior bowing. At the end of 1 month he was walking well with two canes (Fig. 2) and there was good callus. The pin was removed on the fifty-eighth day and the cast on the seventy-fourth day. At this time union was firm, there was 90 degrees of motion at the hip, 45 degrees at the knee, and complete function of the ankle. There was no shortening. At the end of 5 months, the function of the hip and knee was normal (Figs. 4 and 5).

CASE 2. K. K., aged 15 years, Hospital No. 31,157, was admitted with a transverse fracture of the middle third of the left femur with 1.5 inches overriding and posterior displacement of the distal fragment (Fig. 6). There were associated fractures of the left superior ramus of the pubis, the middle third of the right tibia and fibula and the first, second and third right metatarsals. Under spinal anæsthesia the fracture of the right leg was reduced and placed in a non-padded plaster cast. Traction was applied to the fractured metatarsals by wires passed through the pulps of the toes and rubber bands fastened to an outrigger on the cast. The length of the left thigh was restored to normal by traction on the Hawley table. A steel pin was passed transversely through the distal fragment at the level of the adductor tubercle and another in an anteroposterior direction opposite the lesser trochanter. The fracture was reduced by manipulation of these pins and a non-padded spica was applied. Post-reduction roentgenograms showed satisfactory position of all fractures except slight deformity of the distal extremity of the second metatarsal. He was discharged home on the seventh day. He was readmitted for one day on the twenty-fourth day for removal of the wire traction from his toes and repair of the spica which had cracked at the groin. On the sixty-fourth day he was readmitted for 2 days and the casts and pins were removed. There was good union of all fractures but slight lateral angulation of the tibia. When seen again at the end of 4 months he was walking well without support with a slight limp but no pain. The thighs were of equal length but there was ½ inch shortening of the right leg and a right pes planus. Flexion of the right knee was complete. The left knee was limited to 90 degrees. The range of motion of hips and ankles was complete. There was firm healing and good position (Fig. 7).

CASE 3. E. B., aged 42 years, Hospital No. 32,504, was admitted with an oblique fracture at the junction of the upper and middle thirds of the right femur with 1 inch overriding (Fig. 8). Reduction was carried out in the usual manner on the Hawley table under spinal anæsthesia, 2 pins being used and incorporated in a walking spica. She was allowed up on crutches on the third day (Fig. 10) and discharged home on the eighth day. The pelvic portion of the cast was removed on the forty-fourth day. The pins and remainder of the cast were removed on the sixty-fourth day, at which time union was firm. She continued up on crutches. At the end of 3 months she was walking well and had good union with slight anterior bowing. There was 90 degrees range of motion at the knee. Limitation at the hip was considerable as a result of pre-existing arthritis. (Fig. 9).

CASE 4. A. B., aged 29 years, Hospital No. 33,589, was admitted with a fracture of the middle third of the right femur with 2 inches overriding (Fig. 11) and multiple lacerations. The lacerations were débrided and sutured and traction applied to the injured thigh under spinal anæsthesia on the Hawley table. Pins were inserted through both fragments in the usual manner, the reduction completed, and a Boehler spica applied. He was allowed up on crutches on the third day (Fig. 13) and discharged home on the tenth day. Roentgenograms showed satisfactory position (Fig. 12). He was readmitted on the sixty-fourth day on account of a carbuncle near the edge of the ischial pad. The pins and cast were removed to allow incision and drainage. Position was good, but union was not yet firm enough for unsupported weight bearing. Three weeks later the carbuncle was healed and union was strong enough to allow walking on crutches with guarded weight bearing.

CONCLUSIONS

1. A new method of positive mechanical reduction for fracture of the shaft of the femur is presented.

2. Firm, dependable fixation is maintained throughout the period of healing.

3. Ambulatory treatment is feasible, giving the advantages of continuous physiotherapy.

4. Anatomical and functional results in a small series have been excellent.

Since this paper was submitted for publication June 1, 1934, McWhorter has reported the use of a pin through the greater trochanter which is fixed in plaster after reduction of the fracture on the Hawley table. Hawley has also described an apparatus for applying skeletal traction and counter traction to the femur with pins in both fragments. Further experience with the method I have described leads me to believe that it is important to restore the length of the limb before inserting the pins. This will prevent pressure necrosis of the skin about the pins. It is also advantageous to leave the pins free of traction force to allow easy and accurate manipulation.

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THE CLOSURE OF CHRONIC OSTEOMYELITIC CAVITIES BY PLASTIC METHODS

JOHN PRENTISS LORD, M.D., F.A.C.S., OMAHA, NEBRASKA

A SIMPLE serviceable measure for effecting healing in bone cavities is by skin grafting, first used by the writer in 1901 before either saucerization or skin grafting was recommended or popularized (3).

Figure 2 represents a bone cavity in an acetabulum; and the partially excavated head and neck of the femur in a woman 60 years of age, who had had a discharging sinus for 50 years, due to hip disease from infancy. In later years, it had become a large, persistent, residual abscess, because the innumerable curettings and excavations resulted in its progressive enlargement, thus decreasing the possibility of healing the putrid bony cavern. The cavity, which was 7 centimeters deep, was healed by the introduction of plastic skin flaps, and supplemented by skin grafts. The method employed resulted in the permanent healing of this almost life-long problem case (Fig. 2B).

Among the simplest of the defects to be closed following operations for osteomyelitis is the gutter remaining in the tibia due to the removal of the diseased tissue of the anterior and interior portions of the bone after it has been saucerized. The gutter may be long or short. It sometimes extends the whole length of the tibia.

It is represented in the upper third in Figure 4. The common textbook treatment of this condition is to gutter the bone—that is, to excise by saw and chisel the diseased portion and remove sufficient of the cortex to render the cavity more shallow, after which our usual method is to turn in the skin edges to meet at the bottom of this gutter, to cover the bone surfaces, and thus effect early healing and closure.

In some chronic cases in which operation had previously been done, greatly enlarged bones, sometimes covered with extensive exposed scars, had frequently become abraded and ulcerated. Having seen 2 cases of cancer develop from such scars, repeatedly traumatized, I have had a wholesome fear of allowing a workingman to carry such a scar on the surface of his shin. In the case of a greatly enlarged osteomyelitic tibia, many times recurrent (not here illustrated), there was a wide scar area of parchment-like, ulcerated skin, which necessitated wide excision; after which there was insufficient sound skin to fill the unusually wide gutter completely. Therefore, their margins were freely undercut, but still their edges would not meet at the bottom of the gutter without too much tension. Lateral incisions were therefore made and undercut to release the flaps so that the skin margins would coapt. For fastening these skin edges I commonly use carpet tacks of a large size or small nails or brads, after the method of Neuber. Sometimes, however, the bone is so hard and eburnated that an ordinary tack may be broken off, in which case it may be necessary to make small drill holes to receive the brads or tacks. The remaining lateral skin defects after relaxation cuts, if wide, may also require skin grafting, in case they cannot be later coapted by the sutures primarily left untied (Fig. 3A).

Figure 4 represents a persistent cavity in the upper end of the tibia. A large perforating defect extended through the tibia to a sequestrum behind the bone, thus leaving a very large cavity, difficult to fill. Nature will begin the closure of such a large cavernous defect in the interior of the bone by granulations and succeed to a certain point, but there will remain an infected cavity or sinus; persistent sinuses may remain indefinitely. Recuretting fails, because the cavity may have become too large to be healed unaided by nature's processes, and it must be filled by transferring tissue. Where transfer of muscle and other tissue was too difficult and impracticable, I have effected healing in some of these cases as follows: By the incisions as shown in Figure 4A, flaps were made from above and below, which were turned into the excavated area. (Fig. 4B). This technique was used after the cavity had been débrided and lined with granulations. The interior was healed by these skin flaps and a dry cutaneous tract secured by adopting the principle of Beck, which was used by him in the treatment of extensive empyemas. These flaps were turned into the cavity, the skin surfaces facing each other, but not in close contact. Thus a skin-lined tract was secured and the coapted flap margins rapidly healed. The opening having been circular, these margins were readily brought together, and a permanent healing obtained. These procedures reduced the period of hospitalization to a minimum. One such patient had been afflicted for 14 years. A sequestrum in the upper portion on the

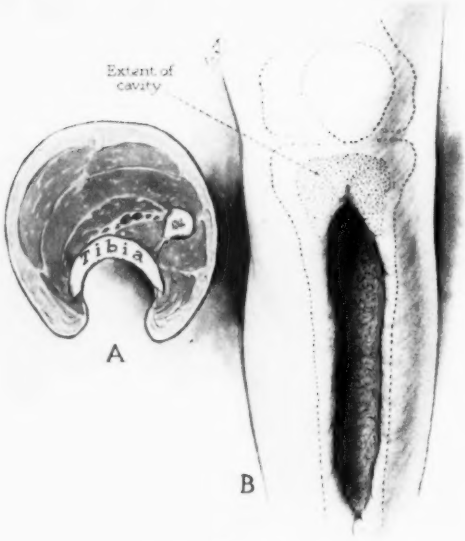

Fig. 1

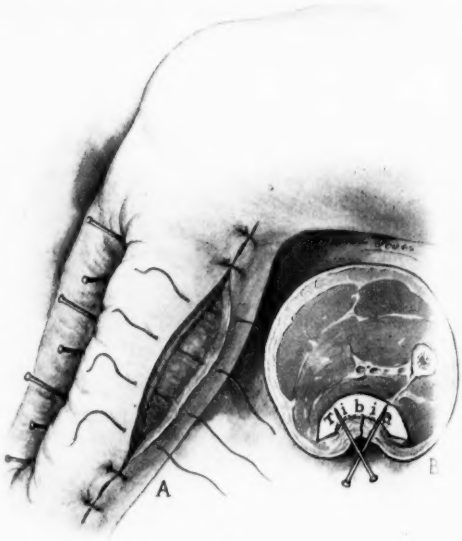

Fig. 3.

posterior surface of the tibia, had left a large tunnel through the bone from eight previous operations, which resulted in successive increases in its size and presented a perplexing problem. The procedures here outlined turned out very well in these cases. Thus seemingly incurable discharging bone cavities were finally and permanently healed in a satisfactory manner. The cavities in the tibia were too high and inaccessible for muscle flaps. The skin-lined tracts (Fig. 4C) gave no inconvenience.

Figure 5 illustrates the preparation of an extensive cavity in the femur, and shows the method of securing the flaps. This technique is not new, but from my observation is little practiced. It is

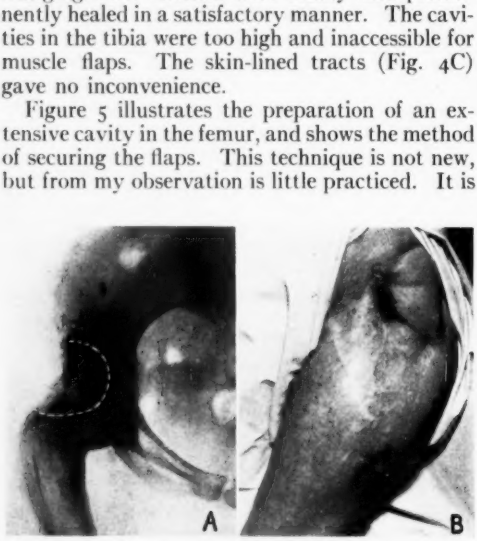

Fig. 2.

my conviction that a person who has had osteomyelitis for 10 or more years, with discharging sinuses and recurrences every year or two, is entitled to an early, expeditious and permanent cure, if such is possible of achievement. The removal of the diseased and infected area in cases of long standing, by careful excision, leaves little chance for contamination and, if infection does occur, it should lack virulence after years of self-immunization. For these reasons, liberties may be taken in making immediate closures. Therefore, it has been my custom to make primary plastic closures by securing tissue as shown in Figure 6A, and turning the flaps of fascia, fat, and muscle secured from the median and lateral thigh surfaces, into the cavity, closing the skin flaps over all in one procedure. (Fig. 5B and C.) In some extensive cases, in which the process involved the whole of the interior of the shaft, as shown in Figure 6A, it has been necessary to turn in tissues from both sides and appose the deep flaps, filling the large cavity completely and closing the skin as in Figure B5.

The following case, a still more extreme one of the type just described, will demonstrate its fuller extent. The process invaded one-half of the femur and involved the condyles to such a degree that the disease extended to the cartilages, and there remained but shells of the condyles. Ankylosis of the knee had resulted from the effects of the disease, which had existed for 24 years. The

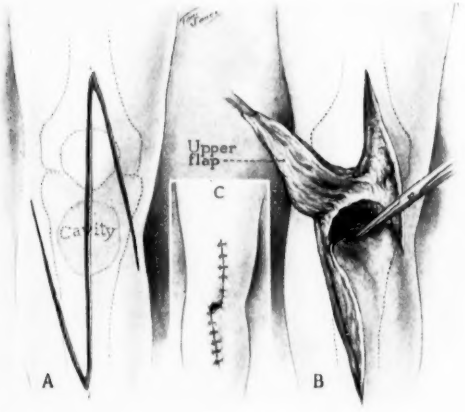

Fig. 4.

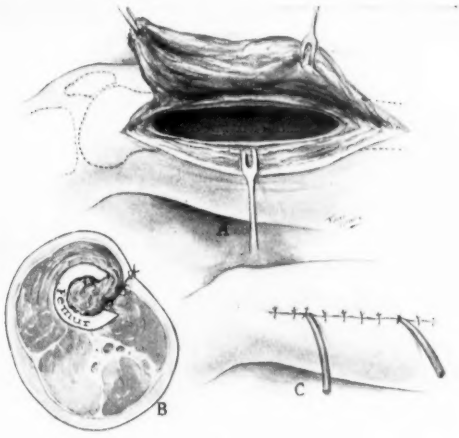

Fig. 5.

apices of the flaps, composed of fat ligaments and underlying soft parts over the ankylosed knee joint, including periosteum over the condyles and tuberosities of the tibia, provided extra length to the flaps of the vastus internus and externus. The extra length of these very long flaps was secured by extending the longitudinal incisions in the skin and ligaments below the knee joint, 1.5 inches; the skin had been reflected and deep flaps of fat, fascia, and periosteum were taken from the lateral surfaces of the ankylosed knee. The femur and condyles had been completely excavated from both sides. These long flaps were then turned from each side into the hollowed lower femur and folded transversely within the cavity of the condyles so as to fill completely this portion of the very large space. The fascia, aponeurosis, and muscles were sutured together, as well as to the wound margins, to prevent their retraction. Thus, by using these large amounts of additional soft tissues to elongate the flaps of the vastus muscles, the enormous cavity in the lower half of the femur and extending into the condyles was filled. Aided by dakinization, delayed or delayed primary union was obtained. Figures 6A and B illustrate the method of closure and Dakin tubes placed as shown in Figure 5C.

In earlier work, I regularly introduced Dakin tubes, and to prevent their immediately becoming clogged with clots which would nullify their efficacy, it was my custom to start irrigation immediately with a 2 per cent solution of sodium citrate and continue it until the Dakin irrigation was established in order to be sure of having patulous tubes. By employing this technique in these cases, a sterile wound was maintained, and healing by delayed or delayed primary union was immediately accomplished, permitting the discharge of the patient in 3 weeks. The patient with the severe condition referred to has remained well. Previous to being treated as outlined, this man had suffered from constantly discharging sinuses, with recurrences every year or two, for a period of 24 years. In a similar case the wound closed promptly after 35 years of sinuses and recurrences. A series of 16 cases somewhat alike have been thus successfully closed by what I have termed "delayed" primary healing.

These operations may vary in degree, according to the extent of the disease, and must necessarily

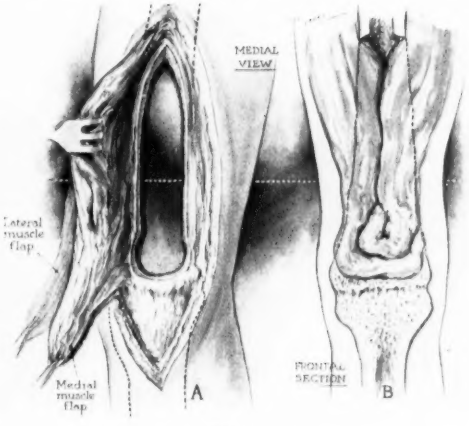

Fig. 6.

vary in their application. I have found dakinization very useful in many cases, but in some of the more chronic cases I have not used it. I have simply coapted the wound rather loosely and left it patulous, and have succeeded without dakinization. It is a matter of judgment as to whether or not one need employ Dakin's solution. In a few cases, hypertonic salt solution has furnished a satisfactory substitute. Needless to say, my judgment has failed me in some cases in which I had become too bold, and had used no antiseptic irrigation.

The plastic closure of osteomyelitic cavities by bone and soft tissue, I repeat, is not new. Experience has led me to use it in the manner described, with variations in selected cases, and the results have been obtained by a primary loose closure of the cavity, rather than by a period of primary dakinization and closing by a secondary procedure.

One of my principal objects in this work has been to limit the stay of the patient in the hospital and secure an early permanent healing.

In the late war, it was found very desirable to treat wounds with Dakin's solution after débridement; and when the field became clean, the plastic work was done as a secondary operation. In civil life, many of our cases are of the chronic form, and repeated operations have been done, the patients having gotten well of the acute disease, but continuing to be harassed by recurrences or by draining sinuses. As previously stated, it is my conviction that such long suffering individuals are deserving of permanent cure and that the operations, therefore, should be radical if necessary to effect it. In the chronic cases, when infection is not particularly to be feared and the patient has become, presumably, relatively immune from the remaining organisms, one may, in a primary operation, do the excavating and plastic closure as one procedure, and secure, at least delayed primary union. In chronic osteomyelitis involving the whole interior of the bone, whether it be of limited or great extent, the cavity may be filled by muscle and fat, and sometimes bone, after thorough removal of the disease. The complete closure and healing of the wound in some cases need not require more than 10 days or 2 weeks.

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OPERATIVE INJURIES OF THE URETER[1]

IRA R. SISK, M.D., F.A.C.S., Madison, Wisconsin

ALTHOUGH a review of the literature reveals numerous reports of operative injuries of the ureters, it is probable that relatively few cases are recorded. This discussion deals briefly with the treatment of some types of such injuries and is based on the observation and treatment of 5 cases.

In considering the question of operative injury of the ureter either from the standpoint of prevention or treatment, the important thing to be kept in mind is the preservation of renal function. Before entering upon a discussion of the treatment of these injuries means of preventing them should be mentioned. The presence of a catheter in the ureter during difficult operations in the pelvis will make it possible easily to avoid the ureter. For many years we have routinely inserted catheters in the ureters of such patients for the Department of Gynecology at the Wisconsin General Hospital, and as a result only one ureter has been damaged (Case 4 of this series). In this case serious pathology was unexpectedly encountered and catheters had not been introduced.

In treating these injuries the genito-urinary surgeon encounters many difficult problems. The variations depend upon the age of the injury, its distance from the bladder, whether one or both ureters are involved, and the type of injury; that is, whether it is completely severed, completely severed with the removal of a segment, partially severed, ligated, or clamped. Suddenly confronted with the problem of dealing with a severed ureter many surgeons adopt the procedure of ligation as the best way out of the dilemma. Embarrassing circumstances are in this way avoided in many cases, but a kidney is rendered functionless. Further, in some cases this treatment results after a lapse of a few days in urine draining from the ureter into the peritoneal cavity, or possibly finding its way to the outside, or infection in the kidney may necessitate surgical interference. The ligation of a cut ureter under circumstances favorable to repair should receive our most severe condemnation.

The method of repairing the ureter when the injury is discovered at the time of operation is for obvious reasons of the greatest importance. However, since the genito-urinary surgeon is rarely present when the injury occurs he will seldom have to deal with this situation. Simple ligation is justified only when the condition of the patient is too bad to permit an operation for repair, for which, of course, more time is required.

A large number of operations have been described for the repair of this type of injury, but all are based on the principles of end-to-end, end-in-end, or side-to-side anastomoses. As this operation will always be infrequently performed, a simple method should be adopted and its principles thoroughly understood, so that when called upon to repair a ureter the surgeon will not have to use a technique with which he is not thoroughly familiar. My first opportunity for repairing a severed ureter came suddenly. Fortunately, a satisfactory repair was made and a normally functioning kidney was obtained (Case 1). Afterward in looking up the subject I found that I had adopted the technique described by Dr. Hugh Young. This technique seems to combine the principles of simplicity, splinting of the ureter, maintenance of caliber, and satisfactory drainage. It is, I believe, the technique of choice for the repair of these ureters with certain exceptions which will be mentioned later. The technique is as follows:

A ureteral catheter as large as will freely move through the ureter without interfering with the circulation is introduced into the ureter and passed into the kidney pelvis. Introduction may be made through a cystoscope or the catheter may be passed each way from the point of injury, the lower end being brought out through the urethra later. Over this catheter end-to-end anastomosis is made with fine catgut, the sutures being passed through the serous and muscular layers. With satisfactory drainage the catheter may be left in place for many days to prevent contraction of the lumen during healing.

Exceptions are to be made when (1) the injury is sufficiently low to permit re-implantation into the bladder, which is preferable; or when (2) a segment has been removed of sufficient length to cause tension. If there is tension satisfactory healing will not occur and implantation into the bowel is the operation of choice.

In old injuries every case is a law unto itself. After appropriate examinations to determine the condition of the kidneys and ureters a satisfactory procedure must be selected. This may be a plastic repair, implantation into the bladder or bowel, nephrostomy, or nephrectomy.

[1]Presented before the American Association of Genito-Urinary Surgeons at Hot Springs, Virginia, May 14, 1934.

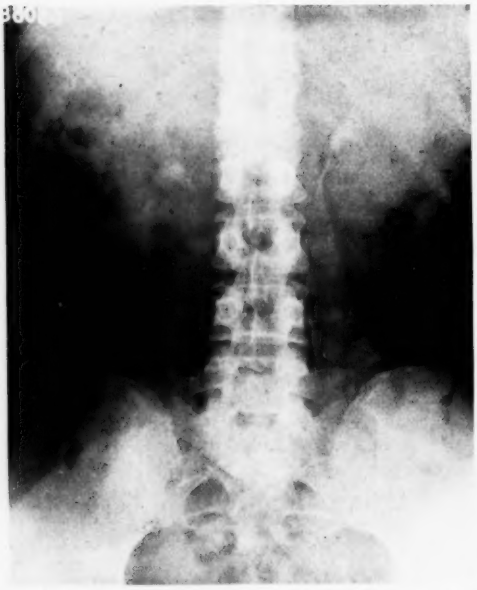

Fig. 1. Intravenous urogram made nearly 5 years after left ureter was completely severed and repaired. The kidney pelvis and ureter appear normal. Case 1.

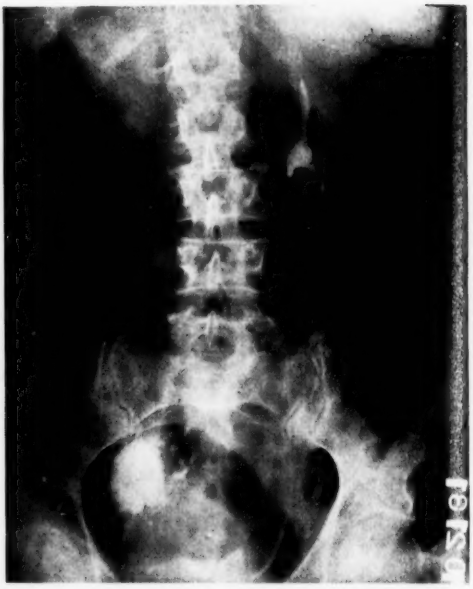

Fig. 2. Retrograde urogram (reversed) showing normal right kidney and extravasation of medium in peri-ureteral tissues on left. This was made more than 2 months after the injury, the ureter having been repaired and the catheter left in the ureter for 15 days after the operation. Case 2.

Case 1. Mrs. J. N., a white female aged 24, was operated upon, April 9, 1929, by a general surgeon for an inflammatory mass of long standing which was located in the left side of the pelvis, having originated in the left fallopian tube. During the course of the operation it was discovered that the left ureter had been severed. Being in the hospital at the time, I was called, and with the wound still open passed a direct cystoscope into the bladder, and introduced a No. 6 catheter into the left ureter. When this catheter reached the point of severance the surgeon introduced it into the proximal end of the ureter and it was passed to the kidney pelvis. The ends of the ureter were then anastomosed—end-to-end—with fine chromic catgut sutures, which were passed through the serous and muscular layers. There was never any leakage of urine at the point of anastomosis. The catheter was removed on the tenth day. Convalescence was smooth and uneventful. The patient was discharged from the hospital on the twentieth postoperative day in good condition. She has been seen at intervals since and has at no time had any symptoms referable to the kidney, and the urine has been normal. She was last seen in February, 1934, when she came for examination at my request. She was free of symptoms, the urine was negative, and intravenous urograms showed normal kidney pelves and ureters.

Case 2. Mrs. B. was operated upon by a general surgeon, August 11, 1931, for the removal of an ovarian cyst. About 4 centimeters of the ureter which was attached to the wall of the cyst were removed. I was called at the time and inserted a No. 12 catheter through a cystoscope as described in Case 1. The ureter was repaired as in Case 1 but with considerable tension due to the removal of a piece of the ureter. On the seventh day a small amount of urinary drainage was noted on the dressing. There was no further drainage, and recovery seemed uneventful. The catheter which had drained freely was removed on the fifteenth day and the patient went home on the nineteenth day with the wound well healed. Ten days later, however, she developed symptoms of frequent and painful urination and the urine was found to contain considerable pus. She was treated for several weeks with bladder lavage and urinary antiseptics, but was returned to the hospital on October 13, 1931, when she had a chill, fever, and pain in the right side of the abdomen. On cystoscopic examination the right kidney was found to be infected with Bacillus coli. No urinary spurt was seen on the left side and though the left catheter apparently passed to the left kidney the urogram indicated that it passed into the peri-ureteral tissues at the point of severance where it was coiled. The proximal end of the ureter apparently healed with occlusion as an intravenous urogram showed no evidence of dye in the left kidney pelvis and ureter.

Case 3. Mrs. H. K., a white female, aged 48 years, had a vaginal hysterectomy and repair of cystocele by a general surgeon on March 24, 1931. Following the removal of the vaginal pack urine was noted from the vagina. From cystoscopic and other examinations it was determined that this was due to an injury to the left ureter. On October 1931, through a left rectus incision the left ureter was isolated, freed, and re-implanted into the bladder. Convalescence was uneventful and the patient was discharged from the hospital on the twenty-first day in good condition. The patient has been seen at intervals since and has been free of symptoms referable to the urinary tract. She was last examined on April 15, 1934, when she was free from symptoms and the urine was normal. An intravenous urogram at that time showed good function in both kidneys and very little dilatation in the left pelvis.

Fig. 3. Intravenous urogram 3 years after reimplantation of injured left ureter into bladder. The pelvis, calyces, and ureter are normal or very slightly dilated. Case 3.

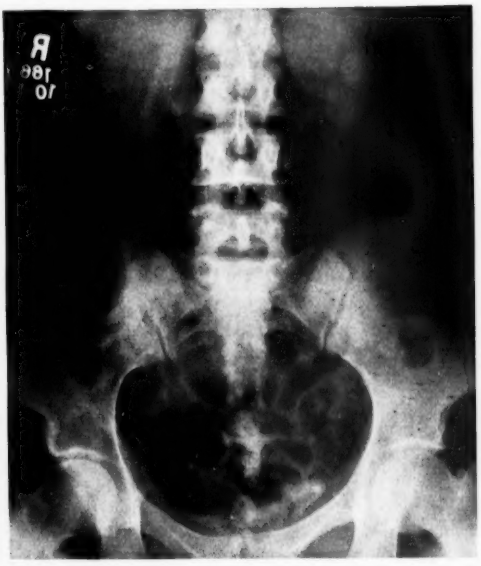

Fig. 4. Intravenous urogram showing the presence of hydronephrosis on the left side which was the side injured. Case 4.

CASE 4. Mrs. C. M., a white female aged 46 years, was operated upon by a gynecological surgeon in November, 1930, for a very large fibroid tumor. When ligating the left uterine vessels a structure thought to be a severed ureter was encountered. It was ligated, the abdominal wound was closed, and vaginal drainage instituted. A few days after operation urine began to drain through the vagina. Four weeks after operation an intravenous urogram revealed a large left hydronephrosis. A left nephrectomy was performed. Convalescence was uneventful. As large aberrant vessels were found going to both poles of this kidney, it is probable that the vessel to the lower pole was at least partly responsible for the hydronephrosis.

CASE 5. A white female, aged 38 years, was first seen on June 24, 1931. She presented a letter from her local physician who stated that he performed an operation in March, 1931, for the removal of cysts from both broad ligaments. The left ureter was cut and repaired by invaginating the proximal end into the distal end. The wound healed, but 10 days after discharge from the hospital severe chills and fever occurred, the wound re-opened and urinary drainage was noted. This continued. When we first saw the patient she was very acutely ill with temperature 103–104 degrees, chills, fever, nausea, and vomiting. There was free urinary drainage through the old abdominal incision. Urine was voided regularly. On cystoscopic examination urine was seen spurting from the left ureter and indigocarmine appeared in 9 minutes. A catheter met an impassable obstruction 3 inches above the bladder. No urine was seen coming from the right ureter and indigocarmine did not appear. A catheter met an impassable obstruction 5 inches above the bladder. A diagnosis was made of complete occlusion of the right ureter, probably the result of ligation, and left uretero-abdominal fistula; part of the urine from the left kidney draining into the bladder and part through the fistula. Operation was performed on July 14, 1931. The left ureter was located and was found to be firmly adherent to the iliac artery for a distance of 1½ inches; this was carefully dissected free. The fistulous tract which passed through the abdominal

Fig. 5. Retrograde pyelo-ureterogram, right. Case 4.

Fig. 6. Intravenous urogram. The left pelvis, calyces, and ureter are moderately dilated. No dye was excreted by the right kidney. Case 5.

Fig. 7. Bilateral ureterograms (retrograde reversed). Catheters could not be passed to kidneys as one ureter was completely occluded and the other was nearly occluded at the point of injury. Case 5.

cavity was dissected from the ureter and the peritoneum was closed. The ureter was then opened above the strictured area and the stricture was widely dilated. Through the opening in the ureter a No. 12 catheter was passed upward to the kidney pelvis and downward through the bladder to the outside. The ureter was sutured and the wound closed with drainage. As a result of the free drainage the patient's condition in a few hours was better than before the operation. The catheter drained freely and in large quantities and there was no urinary drainage on the dressings. The patient died suddenly on the third postoperative day, apparently from an embolus, though this could not be confirmed as autopsy was refused.

There are in the literature many case reports in which it is assumed that a satisfactory result has followed a ureteral repair, but few in which the question is proved by appropriate follow-up examination. In 1929, Curtis reported a case in which a satisfactory result followed an end-to-end anastomosis and stated, "I believe that this is the only recorded case in which end-to-end anastomosis of the ureter has been followed by clinical proof of normal kidney-ureter function." To the successful proven cases we add Case 1, and as further proof of the well known fact that the absence of symptoms following the repair of a ureter does not prove the result satisfactory, we cite Case 2 in which complete healing with occlusion occurred after the removal of an indwelling catheter which permitted free drainage from the kidney for 15 days after the operation.

CONCLUSIONS

1. Normal kidney-ureter function may be obtained following complete severance of a ureter by end-to-end anastomosis over a ureteral catheter.
2. Simple ligation of a severed ureter is contraindicated if the condition of the patient will permit a conservative operation.
3. The absence of symptoms following the repair of a severed ureter does not prove the result satisfactory.
4. In old injuries of the ureter a suitable procedure must be adopted to each case. Re-implantation into the bladder is desirable if possible. Implantation into the bowel, nephrostomy, or nephrectomy may be indicated.

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THE PROGNOSIS OF THYROID CANCER

HOWARD M. CLUTE, B.Sc., M.D., F.A.C.S., AND SHIELDS WARREN, M.D., BOSTON, MASSACHUSETTS

IT is generally recognized by all students of thyroid cancer that considerable confusion exists because of a terminology which varies with each author and because of different interpretations of the histology. We believe that, in the last analysis, histological studies of thyroid cancer are of value only as they benefit the clinical management of thyroid cancer cases. Attempts to associate certain histological pictures with more or less precise clinical findings have been made by pathologists since the days of Virchow. Such studies have proved of definite value in the clinical management of cases of buccal malignancy, bladder malignancy, bone malignancy, and so forth. It is logical to apply these methods to a study of cancer in the thyroid, and we believe that by this method very definite help is provided to the clinician.

It has long been apparent to clinicians that certain types of malignancy in the thyroid were relatively benign, slow growing, and more or less amenable to treatment. It has been equally apparent that other cases of thyroid malignancy were extremely rapid in their course, and entirely uninfluenced by treatment. It is our experience that these clinical types are characterized, in general, by very definite histological anatomy and that the clinical grades of thyroid malignancy fall readily and easily into corresponding histological classes.

Our studies of thyroid malignancy first correlated these findings in 1929 (2) but in 1931 (3) the present method of grouping thyroid malignancy both from the clinical and the histological findings was emphasized. Increasing experience in the method that we discussed in 1931 has demonstrated to us its distinct value. We have repeatedly been able to predict the histology of certain thyroid cancers from our clinical observation of the patient before operation and we have usually been correct in our clinical prognosis of thyroid patients once the histology of the cancer had been presented to us by the pathologist.

We divide thyroid cancer into three clinical groups. In group I we have the patient with low or potential malignancy. In group II we have the patient with definite but not hopeless malignancy, and in group III we have the patient with severe and usually incurable malignancy. Patients can be placed in these clinical groups on the histology of their tumors. Only by this combined histological and clinical grouping can an adequate prognosis be given and a constant background for comparison of results be obtained.

MATERIAL FOR STUDY IN THIS PAPER

In this review there are included 226 cases of cancer of the thyroid in which we have histological sections for study. The great majority of these patients have been operated upon, but in a few of the cases only autopsy material is available. These 226 cases have been seen in the Lahey Clinic up to January 1, 1932. None of the cases occurring in the Clinic during 1932, 1933, or 1934, has been included.

Up to January, 1932, there have been 18 additional inoperable cases of cancer of the thyroid in the Clinic which are not included in this study. Since no tissue was available in these cases they could not be classified histologically. All 18 are now dead.

Many of these 226 cases of cancer of the thyroid have been previously studied and reported upon. Thus, in 1929, Clute and Smith (2) reported 67 cases of malignancy seen in the Lahey Clinic, and in 1931 we (3) made a further study of all thyroid malignancy in the Clinic including those previously reported. The present study includes the cases reported in these two previous papers and such new cases as have occurred in the Clinic up to January 1, 1932 (Chart 1).

A constant follow-up of all cancers of the thyroid seen in the Clinic is maintained. In the group I cases, the least malignant type, only 6 were completely lost. In groups II and III no cases were lost. We have, therefore, a 97 per cent follow-up on the 226 cases of thyroid cancer reported in this paper.

The papers on thyroid malignancy by Wilson, Graham, Pemberton, Wegelin, and Haagensen have well summarized the older literature as well as presented important new facts. It is unnecessary to review again the literature on this subject which these writers have covered.

GENERAL CONSIDERATION OF CANCER OF THE THYROID

Symptoms. The classical symptoms, so called, of cancer of the thyroid are really those of the end stages of thyroid malignancy. A rapidly growing goiter with irregular lobulation, distort-

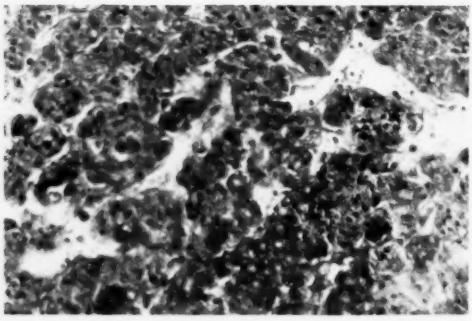

Fig. 1. Fetal adenoma with blood vessel invasion, malignant. No. 4858. ×150.

ing the usual symmetrical shape of the thyroid gland is rarely present until the condition is hopeless. Hoarseness, stridor, choking, inability to swallow, all appear late in the disease as invasion of the larynx, laryngeal nerves, trachea, and œsophagus take place. While we must state these symptoms and recognize that they arise in the final stages of this disease, it is much more important to be familiar with the earliest findings suggestive of thyroid cancer.

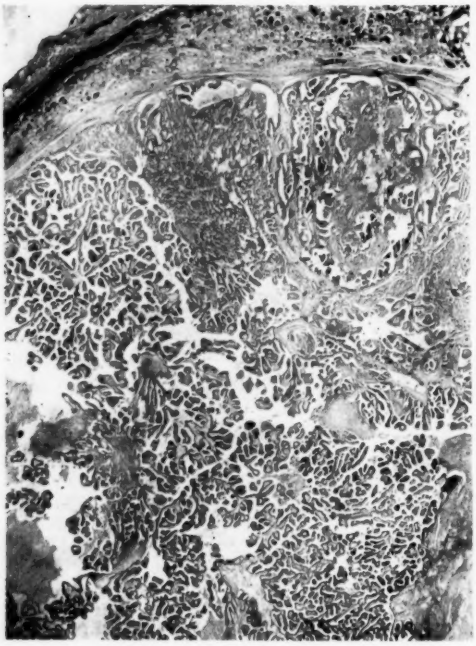

Fig. 2. Papillary cystadenoma, malignant. No. 21067. ×5.

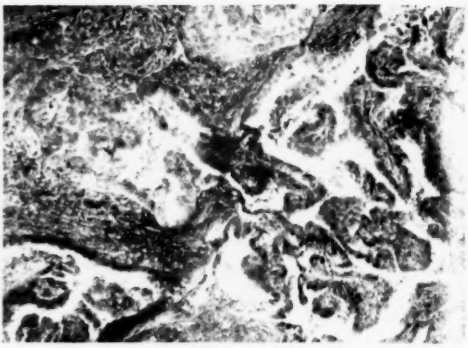

Fig. 3. Adenocarcinoma, papillary type. No. 15[illegible] ×75.

In the early stages of thyroid malignancy, just as in the earliest stages of cancer of the breast, there are no symptoms which are positive and diagnostic. The significant and suggestive findings, however, which point to the possibility of cancer in the thyroid are the slow growth of an

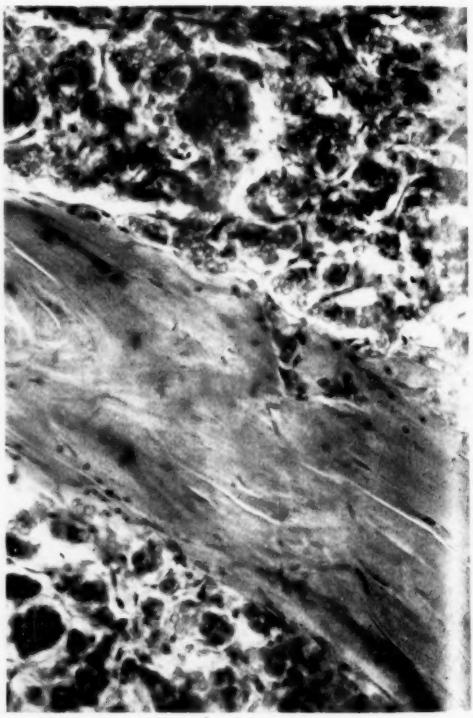

Fig. 4. Adenocarcinoma, alveolar type. No. 23[illegible] ×165.

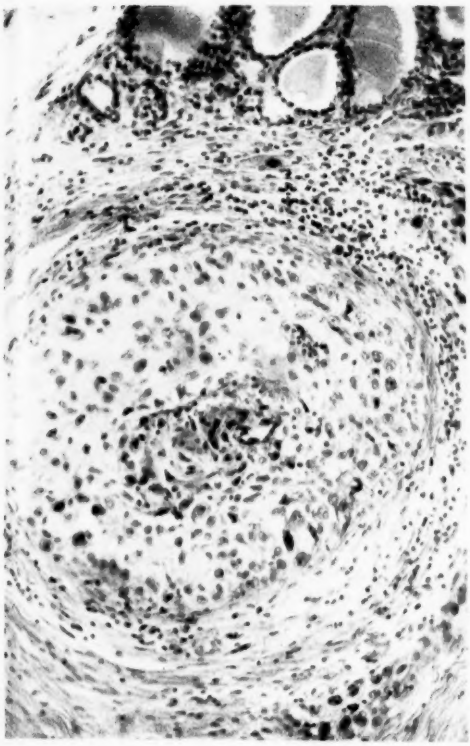

Fig. 5. Squamous cell carcinoma. No. 6003. ×365.

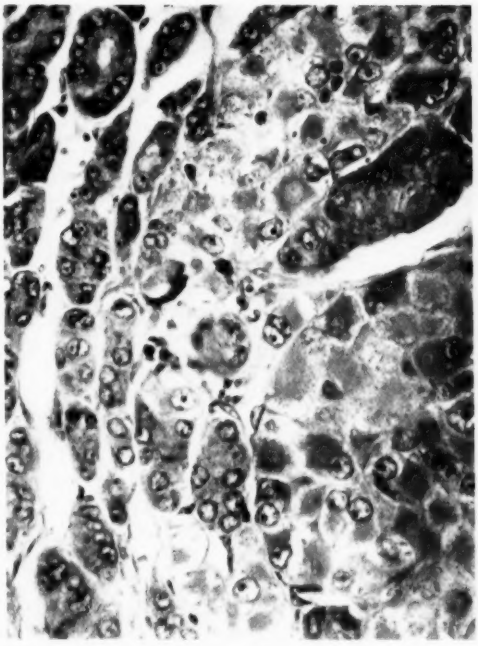

Fig. 6. Carcinoma of Hurthle cell type. No. 21145. ×400.

adenoma over a period of weeks or months. Increasing firmness of the tumor and the occurrence of a sense of pressure in the neck with lack of freedom of its movement on palpation is also seen in relatively early stages.

Clinically, we must differentiate thyroid tumors suspected of malignancy from cases of chronic thyroiditis of the Riedel's type and the Hashimoto type, from cases of acute thyroiditis and from cases in which there has been a sudden hæmorrhage into a benign adenoma.

Riedel's struma is perhaps as frequently mistaken for cancer of the thyroid as any thyroid swelling. Not infrequently the final differentiation of this tumor is made only by histological study of sections. In general, however, Riedel's struma is diffusely and usually bilaterally of a stony hardness but the enlargement of the thyroid is symmetrical. There are, as a rule, no nodules and the superior poles and inferior poles on each side can be felt to be much like those of a normal gland, though enlarged. There is rarely fixation of the tissues in the neck in spite of the hard thyroid tumor, nor is there evidence of recurrent laryngeal paralysis. Local tenderness is often noted in Riedel's struma.

In Hashimoto's thyroiditis or struma lymphomatosa, we have a rare type of firm thyroid enlargement which, like Riedel's struma, is apparently of an inflammatory nature but which is marked by a diffuse lymphoid infiltration of the thyroid and by a definite enlargement of the entire thyroid. Here, also, there is no diffuse involvement of the tissues of the neck, the enlargement maintains the usual contour of the thyroid gland but is firm and hard and very frequently is considered to be a case of chronic thyroiditis. Here the correct diagnosis is generally impossible until histological studies are made.

In acute thyroiditis, the history is usually very suggestive of the type of lesion. In most cases a preceding respiratory tract infection has occurred. Within a few days or weeks a tender enlargement of one or both lobes of the thyroid gland appears. On palpation the thyroid is found to be symmetrically or unilaterally enlarged. The enlargement, however, retains the general contour of the gland. There is very marked and definite

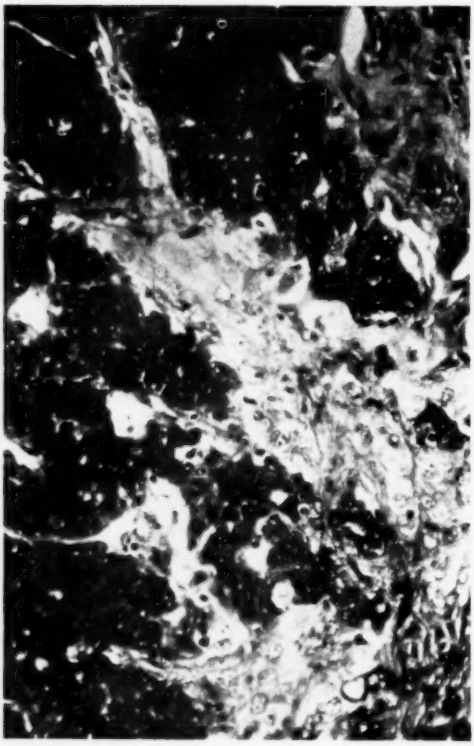

Fig. 7. Carcinoma simplex, compact type. No. 22977. ×170.

local tenderness. Often there is a low fever, and occasionally evidences of hyperthyroidism of mild degree are present.

Hæmorrhage into a thyroid adenoma may produce a most confusing picture. The history, however, in most cases gives the correct diagnosis. The patients may or may not have previously noted a goiter. Suddenly, as a rule following exertion, the adenoma of the thyroid increases definitely in size. This swelling occurs in the matter of a few hours or a day and is associated with marked local tenderness and at times with symptoms of choking and pressure in the neck. The tumor, following hæmorrhage, tends to subside slowly over a period of days or a few weeks. Such a history differentiates at once the firm, hard swelling which is present on examination in these cases as due to a recent hæmorrhage and not to malignant degeneration. Malignant degeneration of an adenoma though it may be rapid, is never a condition developing within a matter of hours or 1 or 2 days.

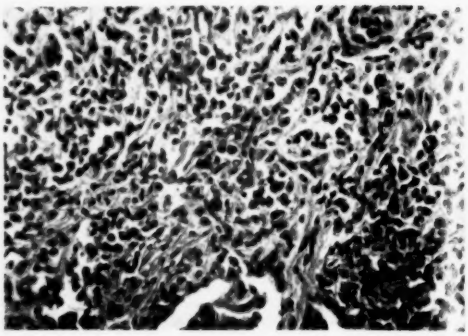

Fig. 8. Carcinoma simplex, diffuse type. No. 18[illegible] ×225.

Thyroid malignancy is suspected, then, because of a firm, hard discrete type of tumor in the thyroid gland, because of recent growth, either slow or rapid, and because of secondary evidences of pressure such as difficulty in swallowing and breathing, and hoarseness. Thyroid malignancy soon leaves the normal contour of the thyroid gland to grow in an irregular and unrestrained manner and to become adherent to adjacent structures. In a few cases, the presence of enlarged lymph nodes near the goiter is suggestive of the presence of malignancy. In rare cases bone metastases may be the first indication of the presence of malignancy in an apparently benign adenoma.

Sex. In 226 cases here studied, 198 were females and 28 males, an incidence of 7 females to 1 male. The incidence of thyroid disease is in general much greater in women than in men. During the past 5 years, 4,779 patients were operated upon for goiter. Of these 648 were men —an incidence of 7 females to 1 male for all thyroid disease. The similarity in these figures is impressive, and may indicate the common origin of cancer in previously diseased thyroids.

Age. Cancer of the thyroid while appearing most commonly in middle life may nevertheless appear at any age (Chart 2). The youngest patient in the series was 9 years of age. Increasing experience with thyroid malignancy has demonstrated to us the great fallacy of thinking that youthfulness of the patient precludes the presence of cancer of the thyroid. Of our patients 8 were 20 years of age or less, and 2 of these have died of cancer, 1 aged 9 and 1 aged 13. Furthermore, it is to be noted from the chart that 39 of our patients (or 16 per cent) were less than 31 years of age, and 77 or more than one-third of all our patients were less than 41 years of age.

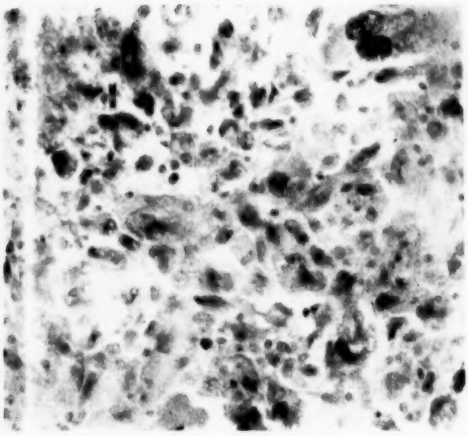

Fig. 9. Giant cell carcinoma. No. 11245. ×365.

The age distribution chart shows that there is a preponderance of group I thyroid tumors in the age of greatest sex activity, the great majority of these patients being 20 to 50 years of age (Chart 3). No such marked association with the active reproductive period is noted in the more malignant tumors of group II or group III which in fact tend to be more common after middle life (Charts 4 and 5).

Previous goiter. It has been stated by different writers on cancer of the thyroid that an adenoma of the thyroid or an adenomatous goiter preceded the malignancy in 90 per cent or more of all cases of thyroid cancer. We have attempted to obtain accurate figures as to the presence of a pre-existing goiter in our group of cases. Such figures, however, are open to a certain amount of question because of fallacious observations by patients. In many cases, however, the pathology of the gland establishes the presence of an antecedent adenoma. We may generalize from this series and say that a goiter was almost invariably present for an appreciable time before operation in the patients of groups I and II. In group III, however, we find that often no goiter was noted longer than a few weeks or months before operation.

Cancer may occur coincidentally with exophthalmic goiter. We have in our records 4 cases of exophthalmic goiter and coincident malignancy of the thyroid gland (1). In these cases it is our belief that the malignancy occurred in a coincident adenoma in the hyperplastic gland but that the hyperthyroidism was related only to the presence of hyperplasia in the otherwise normal thyroid tissue.

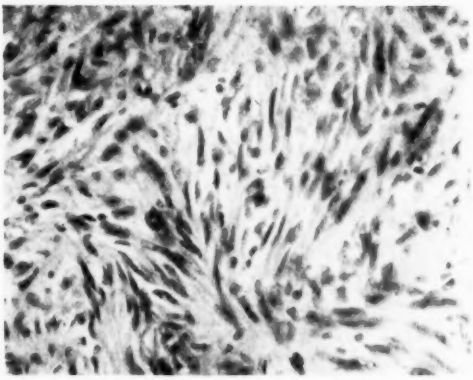

Fig. 10. Fibrosarcoma. No. 18704. ×225.

We have no evidence that hyperthyroidism arises as a result of the activity of malignant thyroid tissue itself. There is, however, evidence that some thyroid malignancies have secretory power. The classical example of function is afforded by Eiselsberg, who reported the development of hypothyroidism in a woman after complete excision of an adenomatous thyroid gland. With the development of a large nodule in the sternum the hypothyroid symptoms disappeared, but on removal of the sternal nodule, which proved to be a metastatic adenocarcinoma of the thyroid, she again became hypothyroid. The presence of active principle in the tissue of struma ovarii has also been well established.

GROUP I. LOW OR POTENTIAL MALIGNANCY

Group I is divided pathologically into 2 classes: (1) adenoma with blood vessel invasion and (2) malignant papillary adenocystoma. We place first the adenoma with evidence of blood vessel invasion (7). The adenoma is of usually undifferentiated type, either embryonal, consisting of simple stalks of thyroid cells without evidence of alveolar formation, usually arranged in a loose œdematous stroma; or fetal, with numerous small follicles, often showing no colloid, with some cords or stalks of undifferentiated epithelial cells (Fig. 1). The stroma in these is also frequently abundant and œdematous with thin walled vessels, so just what we can regard as significant blood vessel invasion is uncertain. In 1,114 adenomata examined by Warren, 3.1 per cent showed varying degrees of invasion of blood vessels and 10 per cent of these died of metastases. Adenomata in which fully developed carcinoma is present are not included in this group, but are classified under the appropriate type of carcinoma.

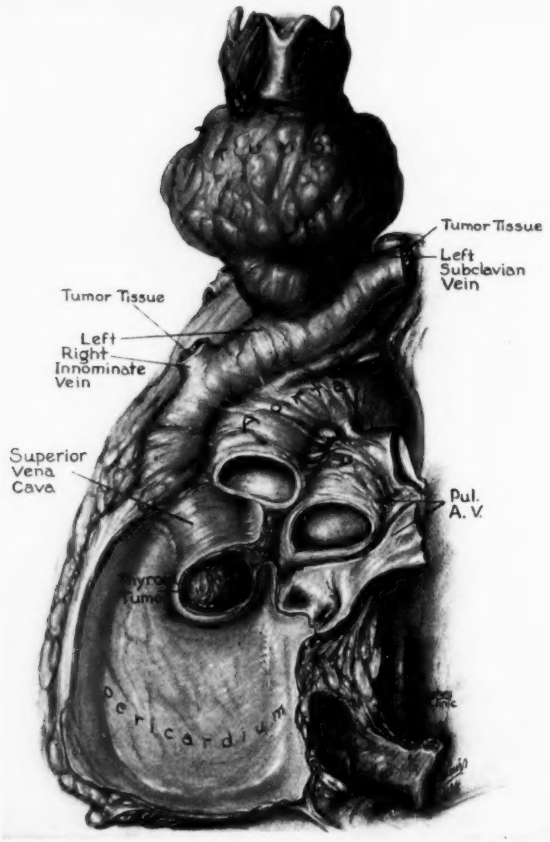

Fig. 11. Artist's drawing of autopsy specimen. Patient aged 60. Goiter 30 months—histologically adenocarcinoma of the thyroid with blood vessel invasion. Note the growth of tumor tissue through the subclavian and innominate veins into the superior vena cava. A similar case was previously reported in 1929 from this Clinic (Reference No. 1).

When grossly demonstrable invasion of vessels is present there is no question as to the malignancy of the tumor. In the group in which there is microscopic evidence of invasion only, there is a 95 per cent chance that the tumor is not actually malignant. However, the appearance of the 5 per cent which metastasize and kill and the 95 per cent which do not is identical. Thus the only safe course is to regard all relatively undifferentiated adenomata showing any evidence of blood vessel invasion as potentially malignant, and those showing gross evidence of blood vessel invasion as definitely malignant. Rarely in this type of tumor is it the metastasis, particularly the metastasis to bone, that first calls attention to the existence of a thyroid adenoma with blood vessel invasion. We have never seen an adenoma metastasize or recur locally without evidence of blood vessel invasion.

It is important in searching for evidence of blood vessel invasion not to be misled by bits of thyroid tissue adventitiously caught in the vessels during the preparation of the sections. Definite evidence of invasion of the vessel wall and of intravascular proliferation is essential. Invasion of the capsule may or may not accompany vascular invasion. Haagensen, in his recent discussion of radiosensitivity of thyroid malignancy, does

CASES OF CANCER OF THYROID TO JANUARY 1, 1932

Histology and Group	Total No.	Followed	Per cent Follow-up	Died cancer	Per cent Dead	With Recurrence	Dead or Recurrence	Per cent Dead or Recurrence	Dead other causes	Alive and well
Group I 68 per cent—Adenoma with blood vessel involvement[1]	99	85	85	3	3	1	4	4	3	78
Papillary adenocystoma[2]	51	50	98	4	7.8	2	6	11.9	8	36
Totals group I	150	135	90	7	5	3	10	7	11	114
Group II 13 per cent—Adenocarcinoma	31	31	100	17	54.8	4	21	68	.....	10
Totals group II										
Group III 19 per cent—Fibrosarcoma	3	3	100	3	100		3			
Epidermoid carcinoma	1	1	100	1	100		1			
Giant cell cancer	11	11	100	9	81.8		9			2
Small cell cancer	30	30	100	23	76⅔	3	26	86		4
Totals group III	45	45	100	36	80	3	39	86	.....	6
Total cases Carcinoma of thyroid	226	211	93	60	26.5	10	70	30.9		

[1] 8 "lost" cases well 3 yrs. and over.
[2] 1 "lost" case well for 3 yrs.

Chart 1. Tabulation of cases in this study showing division into three groups and the relative mortality of these groups.

not speak of this type of tumor as a separate entity.

We regard papillary adenocystomata as malignant if they show evidence of capsule, blood vessel, or lymph vessel involvement. Inasmuch as the excellent study by Moritz carefully distinguishes the various recognized types of papilliferous tumors of the thyroid, we will not attempt to summarize the various forms of tumors of papillary type which may be encountered but will describe here only that type included in this particular group.

The tumor is cystic, grossly capsulated, sometimes with gross evidence of ingrowth into veins. Origin from aberrant thyroid tissue is not infrequent. At times the origin of the tumor may be multiple, although it is very difficult to distinguish between multiple origin and lymph node metastases. On section, contents of the cyst are usually fairly scanty, sometimes serous, and more often blood tinged. Rarely, evidence of calcification is present in some of the papillæ. Under the microscope we see usually a fairly dense fibrous capsule, often invaded in one or more places by ingrowth of tumor tissue, and a tracery of branching papillæ, usually with a fairly obvious stromal cord carrying an abundant blood supply and lined with a single layer of epithelium ranging from cuboidal to high columnar (Fig. 2). Nuclei are not placed uniformly toward the base of cell as in the case of the more delicate papillary projections seen in the follicles in thyroid hyperplasia, but they may occur in almost any position in the cell, often occupying the center. Mitotic figures vary in their frequency although usually on prolonged search some can be found. These, however, are of but little avail as distinguishing the lesion from the epithelial changes of hyperplasia inasmuch as here also mitotic figures may be encountered in the epithelium. We regard invasion of the capsule, invasion of the blood vessels or of lymph channels as being requisite for the determination of malignancy in this type of tumor.

The marked radiosensitivity of this type of tumor is to be expected because of its papillary character. This is a feature usually associated with a good response to radiation in whatever organ the tumor may arise.

The clinical history of the group I patients is at times suggestive of the lesion which is found on histological examination. A goiter had been present in certainly 90 per cent of these patients for months or years before they came to operation, and as a rule the adenoma had shown no rapid change in size. The patients, however, not infrequently state that it had slowly become larger over a period of months or years. This growth should arouse the suspicion of possible malignant degeneration.

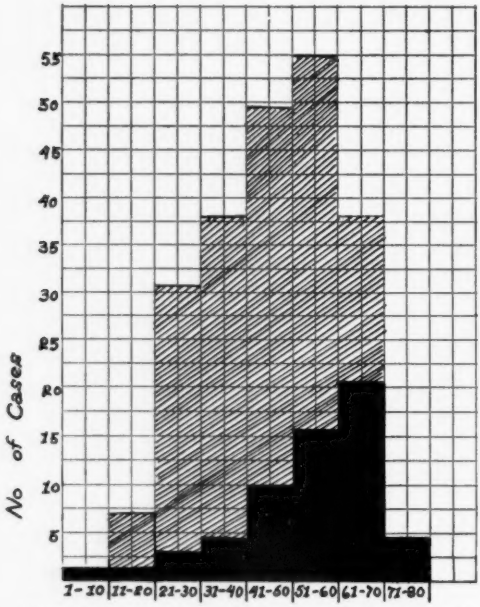

Chart 2. Age distribution of cancers of the thyroid with fatalities of each age decade.

On examination the majority of patients show a discrete adenoma of the thyroid which is often slightly firm but usually is freely movable. Occasionally the adenoma feels quite hard and this strongly suggests malignant degeneration. In a few cases the process has advanced to such a degree that the tumor is large and firm and hard, diffusely attached to the overlying tissues and obviously malignant. This finding, however, is a very unusual one among those cases which are classified under group I.

On clinical examination it is occasionally possible to decide whether the tumor is an adenoma with blood vessel invasion or a papillary adenocystoma. If several nodules are felt along the course of the jugular above the tumor, they suggest very strongly that a papillary adenocystoma is to be suspected.

Of the 99 patients having adenoma with blood vessel invasion, 85 are followed to date, 8 others were followed for over 3 years before they were lost, and 6 could not be traced. Three patients in the 99 are known to have died of cancer of the thyroid, and 1 patient is still alive with a large recurrence present in the neck. Three others have died of causes unrelated to their goiter. Of the 99 patients 86 are well and free from cancer of the thyroid.

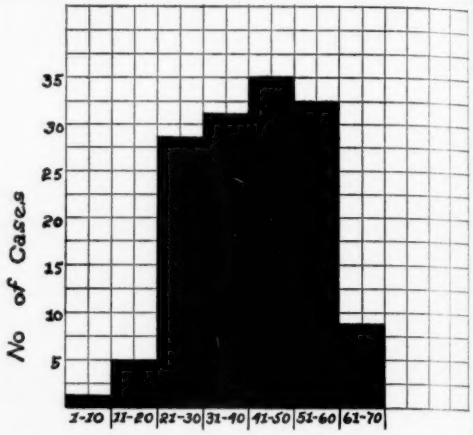

Chart 3. Age incidence of group I cases of thyroid cancer. Note the large numbers in this group occurring in early adult life.

Of the patients having adenoma with blood vessel invasion 89 were females and 10 were males. The age distribution for the cases with adenoma with blood vessel invasion follows closely the age distribution for all the cases in group I as shown in the chart.

The cause of death in 3 patients with adenoma with blood vessel invasion in each instance was local recurrence of the growth with metastases into the mediastinum and into the lungs. In 2 patients death occurred 5 years after the operation, and in the third patient within 1 year. In all 3 patients, however, it was evident for over 3 years that cancer of the thyroid with extensive invasion was present before death occurred.

Of the 51 patients with papillary adenocystoma, all save 1 were followed to date, and the 1 patient when lost was known to have been well 3 years after operation. Of these 51 patients, 36 are well with no evidence of malignancy, and 8 have died of non-related causes. Four patients have died with local recurrences of whom 1 also had a metastasis in the brain. Two other patients in the group have large recurrences present now in the neck which are apparently quiescent under X-ray treatment.

Nine of the papillary adenocystoma patients were males and 42 were females—about 1:5. The age distribution follows that of the group, there having been 1 case (fatal) in a boy of 9 and 6 cases in the sixth decade.

The deaths and recurrences in the papillary adenocystoma cases resemble in their clinical history very much the deaths occurring in patients

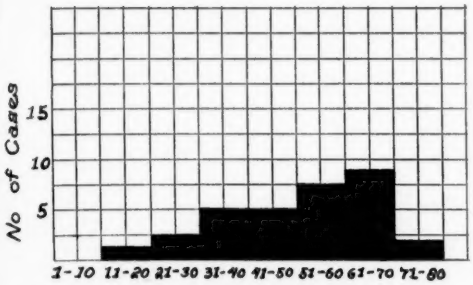

Chart 4. Age incidence of group II cases which tend to be increasingly common in middle age.

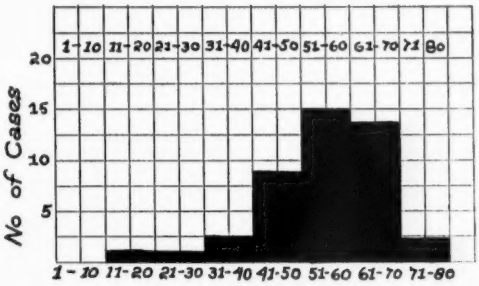

Chart 5. Age incidence of group III cases which like group II occur most frequently at middle and old age.

having adenoma with blood vessel invasion. There were 4 deaths in the patients with papillary adenocystoma from cancer of the thyroid. All of these patients showed local recurrence with slow extension into the surrounding tissue. Two of these patients were inoperable when first seen, yet following biopsy and X-ray treatment, 1 lived 3 years and the other 1 year.

Two of the patients in the group have definite recurrences in the neck with extension into the mediastinum and into regional lymph nodes, one of which has been present, although held quiescent with X-ray, for 3 years, and the other for 4 years. One of the patients with the persistent recurrence was inoperable at the first operation, and only a biopsy was done. Following a year of X-ray treatment, however, it was possible to remove a large proportion of the tumor, and 4 years after this, he was still well though nodules were present in his neck.

Our follow-up studies, then, of these 150 patients of group I type are complete for 135, or 90 per cent. Nine were lost after having been followed and found well for 3 years. Of the 144 cases, 7, or 5 per cent, had died as a result of cancer of the thyroid and 3 others had definite recurrences. This means that 10, or 7 per cent, of the 144 patients followed in group I were dead or had recurrences from cancer of the thyroid. In our previous study in 1931 the mortality of group I cases was 4.5 per cent and it is gratifying to note that as several more years have passed in this group of patients, on further follow up, there is no tendency to increase the mortality from thyroid cancer recurrence. All recurrences or metastases in this group have occurred within a year after operation.

While a great many of the patients in group I have had X-ray treatment as a routine for the first year following their operation, there have, nevertheless, been many other patients who have had no X-ray treatment. It is not apparent to us, therefore, that failure to have recurrence following the removal of the group I type of cancer is due to the use of X-ray treatment. There is no doubt, however, that tumors in group I are quite susceptible to X-ray treatment since we have in several cases seen inoperable tumors shrink to such proportion that their radical removal was possible after X-ray treatment. It is also our experience with the recurrent cases that extension of the growth has been markedly slowed and limited by persistent X-ray treatment after operation.

In all group I patients, recurrence tends to take place along the veins of the neck following the course of the blood stream toward the heart. Nodules, therefore, appear in the mediastinum and in the lung. Local extension by penetration of the capsule by the growth also appears as a rule within a few months after operation if recurrence is to appear. Metastasis of the papillary adenocystoma frequently occurs by the lymphatic stream with involvement of the regional lymph nodes.

For the group I patients then, we may state: first, that all of the patients dying from cancer of the thyroid of the group I type have been patients with clinically obvious cancer of the thyroid which was far advanced at the time of the original operation, and had been present for months or years before operation. All of the patients with local recurrences had extensive growths at their first operation and recurrences were apparent within a year after operation. Only the clinical findings indicate the prognosis in these cases since the histology is alike in all. Finally, in none of the patients that we have followed in this group has any recurrence appeared when the patient was well to clinical and X-ray examination a year or more after the original removal of the thyroid tumor.

GROUP II. ADENOCARCINOMA OF THE THYROID

In group II we place all cases of adenocarcinoma of the thyroid. Here, although the lesion is unquestionably malignant there is some hope of cure and much chance of long relief and palliation by active treatment. In these tumors the usually accepted criteria of malignancy, anaplasia, and invasion of surrounding tissue, are obvious, but the degree of preservation of glandular structure varies considerably. In all of the tumors of this group, however, we expect to find some evidence of glandular structure and alveolar formation. Histologically the group may be readily divided into two forms: the papillary adenocarcinoma and the non-papillary or alveolar.

The papillary form of adenocarcinoma differs from the malignant papillary cystadenoma of group I, by having more than one layer of epithelium over many of the papillary projections (Fig. 3). There is a greater variation of size and shape in the epithelial cells and a definitely greater extent of invasion, not only of the capsule, which is often indistinct or non-existent, but of the surrounding thyroid tissue and muscle as well. Mitotic figures are usually fairly frequent in the cells and tumor giant cells are occasionally encountered. In this type of cancer, distant metastasis is rarely seen but regional lymph nodes are frequently involved, whether by direct extension or by lymphatic metastasis is uncertain.

The alveolar type of adenocarcinoma, as has been brought out by Haagensen and others, presents a fairly wide variety of histological types. These range from forms resembling the more anaplastic of the adenomata (corresponding to the *wuchernde Struma* of Langhans) with predominant alveolar arrangement and not infrequent colloid-containing follicles, to tumors made up of cells barely recognizable as of thyroid origin with, however, some maintenance of alveolar structure (Fig. 4). Even in the less differentiated forms, however, we can distinguish them from adenomata with blood vessel invasion by the slight or absent evidence of encapsulation, by the free invasion of surrounding structures, whether thyroid itself or muscle, and by a distinct degree of anaplasia of the cells, many of which are small, hyperchromatic, and ill-defined. In practically all the carcinomata of this group, evidence of either blood vessel or lymphatic invasion can be found, if search is made. So far as we can judge from illustrations, some of the tumors included in the *wuchernde Struma* we place in the group of adenoma with blood vessel invasion rather than in the group at present under discussion.

One special, though rarely occurring, variety of the adenocarcinoma is the so called Hurthle cell carcinoma (Fig. 6). We have had but one clear cut example of this type although portions of some of our other adenocarcinomata have shown large clear acidophilic cells of the sort considered to be characteristic. The similarity between these cells and certain of the changes in thyroid cells most frequently encountered in involutions following primary hyperplasia led us to follow Ewing and others in considering this tumor as a true thyroid carcinoma rather than as of parathyroid origin as suggested by Eisenberg and others. While admittedly the cells resemble the oxyphil cells of the parathyroid, they appear to us to resemble even more closely the acidophilic phase occasionally encountered in the thyroid epithelium itself.

Because of the diverse character of this group it is rather difficult to lay down any general statement as to its probable behavior in regard to metastasis. Undoubtedly, it would be desirable to separate the extremes of the group, but when the adenocarcinomata are assembled they grade so imperceptibly from one to another that any establishment of a dividing line would be purely arbitrary.

CLINICAL COURSE OF GROUP II CASES

The typical clinical findings in the group II patients are in many respects similar to those present in the group I cases with the exception that the course of the disease is more obviously rapid, more definitely malignant, and more clearly serious.

The usual history of the patient with adenocarcinoma of the thyroid is that a goiter has been present for many years but within a short time, perhaps 6 to 8 weeks, definite and obvious growth of the tumor has occurred. The patient frequently notes pressure symptoms as the tumor enlarges. These may be choking attacks or simple sensations of fullness in the neck. Hoarseness may occur early in these cases if the tumor lies near the recurrent laryngeal nerve.

On examination the usual finding is a discrete adenoma of the thyroid which is hard and firm and may be fixed to the surrounding tissue. It gives the examining finger the definite sensation of malignant tissue localized in a small area. In the more unusual cases the lesion is frankly an extensive malignancy obviously inoperable with wide involvement of all the tissue in the neck.

Three of the patients of group II were males and 28 were females—1:9. Adenocarcinoma is most commonly a disease of middle and late

life as is seen from the chart. It is important to note, however, that examples of this type of tumor may be seen in youth.

In the group II cases there were 31 patients all of whom have been followed to date. Seventeen have died of cancer of the thyroid while 4 others show definite evidence of recurrence. In other words, 54.8 per cent of the patients in this group have died of cancer of the thyroid and 68 per cent have died or have a recurrence of the disease. Ten are alive and well with no evidence of trouble.

In 9 or over half of the 17 fatal cases in group II minor operations such as biopsies alone or tracheotomies or decompressions of the prethyroid muscles were done at the time of operation, and death occurred within days or weeks of this procedure. In 3 of the 8 remaining cases, radical removal of the obviously malignant tumor was followed by rapid recurrence and death. In 5 of the patients, however, radical operation plus X-ray treatment seemed definitely to delay the fatal results, death not occurring until some 2 to 5 years later.

Recurrences in these fatal cases were noted most commonly along the jugular vein and in the superior mediastinum (Fig. 11). Metastases to the lungs were frequent. In 1 patient metastasis was apparently found in the pancreas at an abdominal operation 2 years after the removal of the adenocarcinoma from the thyroid. At this time no evidence of local recurrence was noted but sections from the pancreas were consistent with adenocarcinoma of the thyroid.

Four of the patients in group II are still alive with definite recurrences of their malignancy. In all 4, examination of the neck shows large, hard, firm, fixed masses extending downward into the superior mediastinum. In 2 of these patients X-rays of the chest show definite metastatic nodules present in the lungs. All of these patients have had intensive X-ray treatment, and their recurrences have been known to be present for 6 years, 5 years, 4 years, and 3 years, respectively. In 3 other patients, recurrent nodules which had become localized and more or less discrete following long X-ray treatment, have been removed at second operations. In all of these patients, however, further recurrences have been noted following this secondary removal.

The 10 patients who are well have gone from 3 to 9 years with no evidence of difficulty.

When we reported on cancer of the thyroid in 1931, we found that the mortality in group II for the adenocarcinoma cases was 23.9 per cent. With longer follow up of these same patients and others of this group, however, we note that the mortality now is more than double, 54.8 per cent. It seems very clear that adenocarcinoma of the thyroid is a relatively slowly growing tumor; nevertheless, a most serious one since recurrences and deaths from cancer occur years after an apparently successful removal of the growth.

It is apparent that X-ray treatment has a very salutary effect upon these adenocarcinomata and we believe that it has added to the longevity of patients who had obvious recurrences of the tumor. Furthermore, in other cases, inoperable tumors have become operable after intensive X-ray treatment. It is not apparent, however, that the further removal of these collections of malignant tissue that have become more discrete following X-ray treatment has been followed by any cure of the disease. As time goes on in these patients, brawny induration and thickening of the neck with deeply placed malignant nodules have always been present. Haagensen reports a 71 per cent primary regression with radiation in cases of group II and a mortality of 57 per cent.

GROUP III. HOPELESS MALIGNANCY

We include in group III the most highly malignant tumors. These we have subdivided into 4 classes: the squamous cell or epidermoid carcinoma; the carcinoma simplex or small cell carcinoma with evidence of alveolar formation which may be either compact or diffuse; the giant cell carcinoma, often called carcinosarcoma or giant cell sarcoma; and the fibrosarcoma, which is extremely rare. We do not include in the group the lymphosarcomata. We have yet to see a lymphosarcoma giving clear cut evidence of primary origin within the thyroid in spite of the frequency with which lymphoid infiltration of this organ is seen. In general we believe most of the so called primary lymphosarcomata of the thyroid to be small cell carcinomata of the diffuse type. All the tumors of group III are characterized by marked anaplasia, by frequent invasiveness, by a considerable degree of mitotic activity, and by relatively free metastasis.

Although the clinical course of the group III cases is decidedly uniform, the pathological anatomy is varied. In the class of the squamous or epidermoid type of carcinoma, our only case gave evidence of origin from the thyroid stalk and may represent development from an embryonic rest or possibly from metaplasia of persisting thyroglossal duct epithelium. The presence of definitely keratinized cell membrane, intercellular bridges, and frequent formation of epithelial pearls leaves no question as to the nature of this tumor (Fig. 5).

The second type of tumor in group III, the small cell carcinoma or the carcinoma simplex, shows no evidence of alveolar formation, thus distinguishing it from tumors of group II. The type cell is often small, polyhedral, and ill defined varying markedly in its relation to adjacent tumor cells and to the stroma (Fig. 7). Usually it has a large hyperchromatic, although occasionally vesicular, nucleus. Mitotic figures vary a great deal, sometimes being very frequent and other times rather rare. Invasion of the surrounding tissues is always marked, and blood vascular or lymphatic invasion practically always demonstrable. In the compact type of small cell cancer the epithelial cell masses occur in large or small clusters, closely approximated with one another, with intervening bands of stroma. This structure is usually maintained in the metastases. In the diffuse type (Fig. 8) similar cells are scattered through the stroma, either occurring singly or in clusters of 2, 3, or 4, as seen in the section. The cytoplasm is indistinct and ill defined, the nucleus prominent and hyperchromatic. Often the nucleus is practically all of the cell that can be seen. This, coupled with the irregular scattering of the cells through the tissues, is undoubtedly strongly suggestive of lymphoblastoma. However, we feel the nucleus to be distinctly of epithelial type and the tendency to clumping of the cells in some areas to be suggestive of carcinoma rather than lymphoblastoma. Metastases of this type of tumor are widespread and rarely have we seen a very definite epithelial type of growth in the metastases. As further aid in differentiating these tumors from lymphosarcoma, the response to radiation is of value. In marked contrast to the radiosensitivity of tumors of the lymphoblastic series, this particular type of thyroid carcinoma is, in our experience, absolutely radioresistant. Moreover, in the lymph node metastases the epithelial character of the cells, and the manner of growth clearly distinguish the lesion from lymphoblastoma.

As to the specificity of type of the giant cell carcinoma of the thyroid there is no question. Here again, however, there is dispute as to the exact nature of the tumor cells. We regard them as epithelial. This point was clearly demonstrated recently by Smith. This tumor often presents one of the most bizarre pictures encountered in oncology. Almost every conceivable size and shape of cells with marked variation of nuclear structure are seen (Fig. 9). Mitoses are frequently present and often are atypical to a startling degree. There is a strong tendency for rather plump spindle cells to predominate, although at times polyhedral cells grouped compactly or even in pseudo-alveolar form surrounded by stromal bands are the outstanding feature of the growth. We have not succeeded in demonstrating fibroglia fibrils in relation to any of the spindle cells of this group.

In the last type, the fibrosarcoma, we have three examples. These are apparently perfectly typical fibrosarcomata. Their cells form both fibroglia fibrils and collagen and tend to be fairly uniform, elongated spindle cells arranged in strands, with an elongated nucleus of a somewhat vesicular type (Fig. 10). The character of the cells is fairly uniform throughout the tumor and only by its definite origin in the thyroid could it be differentiated from a typical fibrosarcoma of soft parts.

CLINICAL COURSE OF GROUP III CASES

The typical history in a group III cancer of the thyroid is often sufficient to give a definite suggestion of the type of lesion which is present. In most instances a goiter suddenly appears in middle or late life and rapidly increases in size. In some cases, a goiter has been present before attention was attracted to it by its rapid growth, but this is unusual.

Accompanying the growth of the tumor in the neck are pressure symptoms. These vary from a feeling of discomfort over the swelling with a sensation of pressure on swallowing to actual choking and dyspnœa.

The rapidity with which group III tumors increase in size is often amazing. In many instances a tumor 10 centimeters in diameter is present a few weeks after the first small swelling appeared. This finding is especially common in the giant cell cancers.

On clinical examination the findings which the history suggests of a rapidly growing malignant tumor are usually confirmed. The mass is asymmetrically placed and it is firmly and widely attached to the thyroid gland. One readily gains the impression that the tumor has involved the whole lobe of the thyroid and is not just attached to it or growing in it. Very often the tumor is adherent to the structures overlying the thyroid gland and, therefore, can be but slightly moved. Glands may at times be felt along the jugular vein and chest X-rays may show metastases in the lungs at an early stage in the disease.

As will be seen from the chart, these patients are most frequently at middle life and beyond, regardless of the subtype of group III to which they may belong. All of the patients having small cell cancers were at middle life or over save

one boy of 13 and a woman of 25. All save two of the patients with giant cell cancer were 60 or more years of age and these two were 36 and 54 years, respectively. In the 45 patients in group III only 6 were males and curiously enough 5 of these 6 had small cell cancers while the other one had an epidermoid cancer. All the 11 giant cell cancers occurred in women.

There were 45 patients, or 19 per cent, of this series of 226 cancers of the thyroid who fall into our group III both by their histology and by their clinical behavior. All of these 45 patients were followed to date. Thirty-six, or 80 per cent, have died of cancer of the thyroid. Three other patients now have serious recurrences so that 39, or 86 per cent, of the group III cases are either dead or dying of cancer of the thyroid. In 1931, we reported the mortality of this group as 82 per cent. It would seem, therefore, that the same serious outlook prevails for these tumors with the treatment we have used.

It had been our plan up to January 1, 1934, to do as radical an operation as was possible in each of these cases and to follow this with X-ray treatment. Radium therapy has not been used in these cases up to January 1, 1934, but since that date we have added the use of radium to the operation and X-ray treatment. It is our plan to follow the results of this additional therapy closely and to report upon it at some future time.

In general, X-ray therapy has been very disappointing in the group III cases. In most instances it has not affected the rapidity of the growth of the malignant tumor in spite of the relatively undifferentiated type of cells involved. In only 2 or 3 instances has it been apparent to us that X-ray treatment has checked or controlled the rate of growth. In only 1 case has an inoperable mass been reduced appreciably in size by X-ray treatment.

In 21 or nearly half of the cases in group III only palliative operative measures could be done. These consisted of biopsy only or biopsy plus decompression of the pretracheal muscles or tracheotomy. The very low operability of the group III cases is further evidence of the serious nature of the malignancy since many of them were seen within 2 months of the onset of the tumor.

The recurrences and metastases in the group III cases are seen early in the postoperative course. Local recurrence almost always is present and this tends to spread widely through the neck and into the mediastinum. In 1 patient, the growth spread in a few weeks from the neck into the right axilla. In another patient there was no evidence of any trouble for 4 years after operation when multiple metastases appeared in the spine. In another case of small cell thyroid cancer, retroperitoneal metastases occurred 4 years after the thyroid operation. Late metastases, however, are not common in group III cases. In most instances, recurrences and metastases appear within a few weeks after operation and the course is rapidly fatal.

More than half of the deaths in the small round cell cancer cases occurred within 9 months of operation. There were, however, unexpected examples of longevity in some since 3 patients lived 3 years before dying of cancer and 6 lived over 1 year. Furthermore, in 2 patients no recurrence appeared until 5 and 6 years after operation. Rapidity of growth and recurrence was especially notable in the giant cell cancers where the average length of life after operation in the 9 fatal cases was 4 months and the longest period 8 months. Similar rapidity of fatal endings occurred in the 3 fibrosarcomata and the 1 epidermoid cancer case.

The prognosis for the group as a whole appears to be hopeless with the therapy we have used. The few patients who in our previous studies appeared to be progressing favorably have developed late metastases and died. Of the 6 patients alive and well, 2 had giant cell cancers 2 and 5 years ago. Four had small cell cancers and have lived 2, 4, 5, 10 years, respectively, since operation. This low survival rate graphically shows the serious prognosis of group III thyroid cancers, and we believe that it is this particular group of cases that has given cancer of the thyroid its more or less justly deserved bad name.

SUMMARY AND CONCLUSIONS

1. Cases of thyroid cancer, in their clinical course, fall readily into one of three groups: group I, with low or potential malignancy; group II, with definite but not hopeless malignancy, and group III, with severe and usually incurable malignancy. Clear cut histological findings also mark out these clinical groups.

2. By this combined clinical and histological grouping of thyroid cancer an adequate method is at hand for estimating the prognosis in any case.

3. This paper reports the results in 226 cancers of the thyroid seen in the Lahey Clinic up to January 1, 1932. All but 6 have been followed.

4. The symptoms and diagnostic features of cancer of the thyroid in general are reviewed.

5. Group I cancers of the thyroid are those of low or potential malignancy. Histologically these patients have either adenoma with blood vessel invasion or papillary cystadenoma with blood

vessel or capsule invasion. Seven per cent of these patients are dead of thyroid cancer or have a recurrence. No death or recurrence has occurred in this group in any case free of trouble for a year after operation.

6. Group II cancers of the thyroid have clear cut, definite malignancy for which there is some hope of cure and much chance of long relief. Histologically all are adenocarcinomata of the thyroid. The mortality of the group, however, is 55 per cent, many fatalities having occurred from cancer years after the original operation.

7. Group III cancers are clinically the most hopeless type. Histologically this group contains the squamous cell cancers, the small cell cancers, the giant cell cancers, and the fibrosarcomata. All these tumors are of rapid growth, occur most frequently in middle and later life and usually are rapidly fatal. The mortality of the group III cases is 80 per cent and most of the deaths in this group occur within a few months of the operation.

8. The treatment used in this series of 226 cases has been operation and X-ray radiation. Group I cases are highly sensitive to X-ray radiation. Group II tumors may regress considerably under X-ray which in many cases may prolong life for years. Group III tumors show almost no improvement with X-ray radiation. Since January, 1934, we have supplemented surgery and X-ray radiation with radium therapy in certain cases. This added measure of treatment will be the subject of a later report.

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AN ASEPTIC URETERO-ENTEROSTOMY[1]

EDGAR J. POTH, PH.D., M.D., SAN FRANCISCO, CALIFORNIA
Fellow in Medicine of the National Research Council

ALTHOUGH the Coffey-Mayo technique for the implantation of ureters into the bowel is frequently productive of good results clinically, it is fraught with the ever present danger of infection. The success of this procedure depends upon establishing a check-valve in the distal course of the ureter. This is accomplished by imbedding the ureter between the muscularis and submucosa of the bowel for several centimeters just proximal to its ultimate opening into the colon through the submucosa and mucosa. By applying the principle of the check valve, Coffey succeeded in eliminating dilatation of the urinary tract by the intermittently elevated intra-intestinal pressure.

To insure a properly functioning check valve, however, healing in the absence of infection must occur between the implanted segment of the ureter and the bowel. Only under such circumstances will a minimum of fibrosis and deposition of scar tissue result. A stiff, indurated, non-flexible, intramural channel cannot be readily compressed by an increase of intra-intestinal pressure and will not result in a correctly functioning check valve. Therefore, every permissible safeguard must be employed to prevent infection and secure perfect healing.

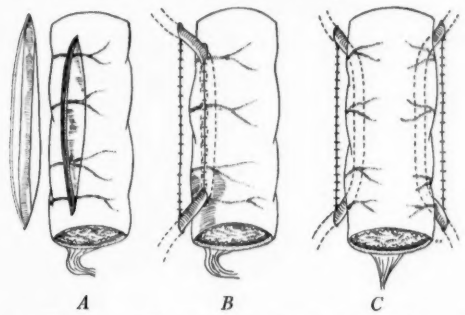

Fig. 1. First stage of operation. *A*, Ureter dissected out. Bed in bowel wall has been prepared near the mesenteric border and down to the submucosa. Blood vessels not disturbed. *B*, Posterior parietal peritoneum has been closed. Ureter imbedded, a Cushing stitch of plain No. 0 catgut being used. Note that the bed is not completely closed; submucosa shows at each end. *C*, Anterior view; showing both ureters implanted without catheters. *D*, Cross section of *C*.

The operative technique for performing ureteroenterostomy to be described fulfills the following requirements: (1) complete surgical asepsis, (2) healing in a clean anastomotic bed, and (3) assurance of an opening between the ureter and bowel.

OPERATIVE TECHNIQUE

The operation is divided into two stages. The first stage is concerned with imbedding the intact and uninterrupted ureters in the wall of the bowel between the muscular layers and the submucosa. At the second stage the ureters are interrupted distally, openings are made between the ureters and the bowel, and the bladder is removed.

First stage of the operation. The first stage is readily followed in Figure 1. The ureters are dissected free for some 10 centimeters, and the posterior parietal peritoneum is closed. The beds to receive the ureters are prepared in the same segment of bowel. Two incisions about 6 centimeters long, extending longitudinally along the bowel approximately 1½ centimeters on either side of the mesenteric attachment, are made through the serosa and longitudinal muscle fibers. The cut edges are separated by blunt dissection by means of the handle of the scalpel. Any remaining circular fibers are carefully divided without injury to the underlying vessels and submucosa. It is not necessary to place both implants in the same segment of the bowel, but since no damage is done to the blood vessels, there is no contra-indication to doing so. In this experi-

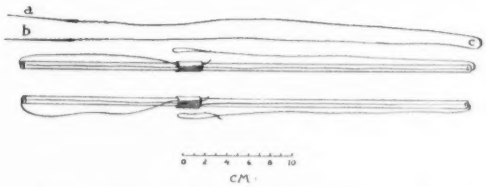

Fig. 2. Glass shields and cutting element. The glass tubes are threaded with silk sutures having loops tied in the free ends. The loop will be hooked over the end of the cutting wire to facilitate hauling it through the glass shield. *a* and *b*, Stiff, blunt darning needles. *c*, is a length of "advance resistance" wire,[1] No. 24 B & S gauge.

[1]The "advance resistance" wire is a nickel copper alloy wire manufactured by the Driver-Harris Co., Morristown, New Jersey. This wire is well adapted to the procedure outlined because it is flexible, has sufficient tensile strength when hot, and does not become brittle after heating. The electric current is adjusted to give a low red heat, which is satisfactory cutting temperature.

[1]Presented before the San Francisco County Medical Society May 29, 1934. From the Department of Surgery, Stanford University School of Medicine. This study was aided by a grant from the Fluid Research Fund of the Rockefeller Foundation.

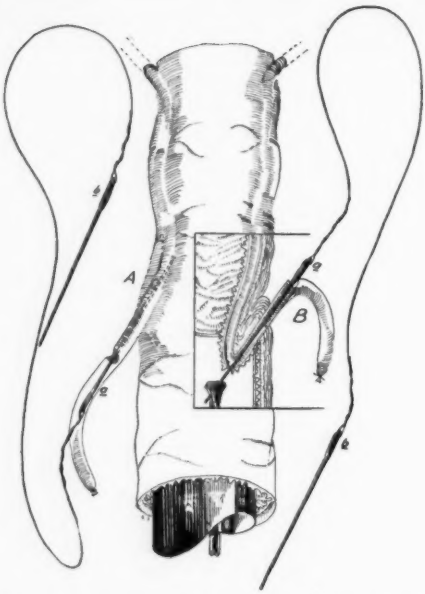

Figure 3. Second stage of operation. Bladder removed; proctoscope inserted. *A*, Needle *a* has been inserted into the ureter and up to a point determining the proximal end of the opening between the bowel and the urinary tract. *B*, Shows needle *a* having been thrust through the visceral wall and grasped by an alligator clamp in the proctoscope from below.

mental work on dogs, all implants have been made at the same level without any evidence of circulatory embarrassment. A single, continuous Cushing suture of plain No. 0 catgut is used to imbed the ureter, the sutures passing into the submucosa. Sufficient tissue is included in the suture to approximate about 3 millimeters of serosa on each side. It is important to leave submucosa of the bowel showing at each end of the bed for a distance of approximately ½ centimeter (Fig. 1*B*). This prevents sharp kinking and compression of the ureters. Urine flows freely within 3 to 4 days and ureteral catheterization is unnecessary. No attempt is made to extraperitonealize the ureters distal to the implant. Omentum is placed over the implants without suturing, and the abdomen is closed.

Second stage of the operation. After 3 weeks the abdomen is reopened. The ureters distal to the imbedded segments are isolated, clamped, and divided at a convenient distance beyond their exit from the bowel wall. The bladder is removed, and the bladder neck cauterized with phenol and closed. At this point a proctoscope is introduced

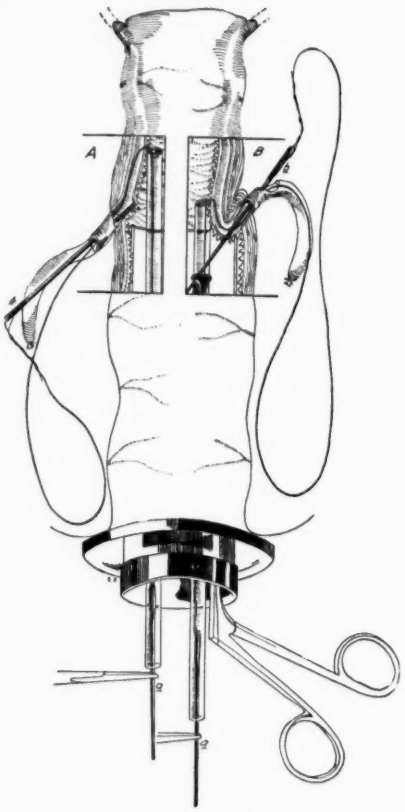

Fig. 4. Second stage of operation. *A*, Half the length of the wire has been pulled through, a shield of glass tubing has been placed over the wire, and needle *a* has been clamped to prevent slipping. Needle *b* is inserted into the ureter with its point just inside the distal junction of ureter and bowel. *B*, Needle *b* has been thrust through the visceral wall and grasped in an alligator clamp.

into the rectum and inserted until the end is opposite the distal junction of the imbedded ureter with the bowel wall (Fig. 3). An opening is made into the lumen of the distal segment of ureter about 2 centimeters from its exit from the bowel wall, and needle *a* of the cutting element, illustrated in Figure 2, is inserted up to the level at which the opening between ureter and bowel is to be made (Fig. 3*A*). With the edge of the proctoscope acting as a fulcrum the needle is rotated forward so as to invert the bowel wall, buried ureter, and needle into the open end of the proctoscope. This accomplishment is evident to the operator from the abdominal cavity, and it is plainly visible from below through the proc to

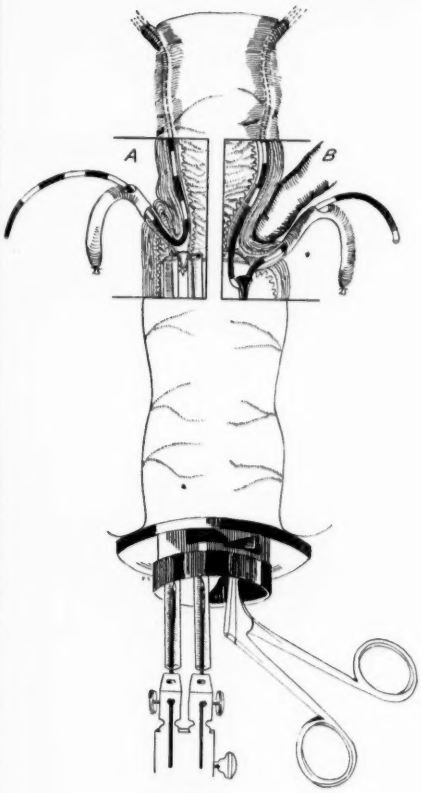

Fig. 5

Fig. 5. Second stage of operation. *A*, Shows entire cutting element in place with glass shields over both legs of the wire, the cautery handle attached, and the protecting

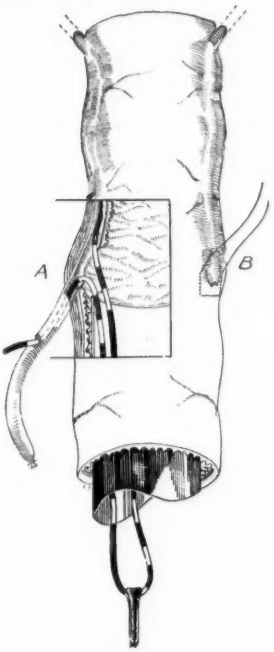

Fig. 6

Fig. 6. Second stage of operation. *A*, Shows the catheter being withdrawn through the proctoscope to obviate soiling of the abdominal field. *B*, The distal segment of ureter has been divided against the bowel wall, the small blood vessel coursing along the ureter has been tied, and a fine suture has been placed to invert the raw area in the bowel wall.

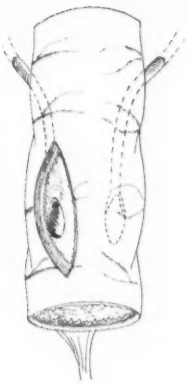

Fig. 7

Fig. 7. Operation completed. A section has been removed to show more clearly the opening into the bowel.

scope. Needle *a* is now thrust through the visceral walls, grasped with an alligator clamp in the proctoscope from below, and drawn out until half the length of resistance wire is pulled through. One of the shields of glass tubing is slipped over the wire as illustrated in Figure 4. Needle *b* on the other end of the cutting wire is put into the ureter and pushed through the visceral walls just inside the distal junction of ureter and bowel (Fig. 4*A* and *B*). This needle is picked up and drawn out of the end of the proctoscope. At this point care must be taken to avoid crossing the legs of the resistance wire which would result in an electric short circuit later. A shield of glass tubing is placed over this leg of the wire and, after the electrical cord to the proctoscope light has been disconnected, both needles are attached to the cautery (Fig. 5*A*). The ureter is catheterized as indicated. By this procedure urine will not pass over the hot wire, and the urethral wall and bowel opposite to the cutting element will be protected by the interposed catheter. Gentle traction on the handle of the cautery pulls the visceral walls into the open end of the proctoscope (Fig. 5*A*). The circuit is closed, and the cauterization is completed in about 15 seconds, whereupon the cutting instrument can be drawn from the proctoscope. The wire should be examined to determine that it is intact. Should it have broken, a second cutting element can be introduced and the entire procedure repeated. However, ordinary care will make this unnecessary.

The bowel wall, overlying the opening, is inverted into the proctoscope with digital pressure, and the distal portion of the ureteral catheter is pushed in to project into the proctoscope as a

loop (Fig. 5*B*). The length of the cauterized opening is clearly visible from below. The catheter is removed through the proctoscope to obviate soiling of the operative field (Figs. 5*B* and 6*A*). The distal segment of ureter is divided closely against the bowel wall, the ureteral vessel ligated, and the raw area covered by a single suture in the wall of the colon (Fig. 6*B*).

Immediately following the completion of this procedure on the one side, it is repeated on the opposite ureter. There is practically no bleeding, and urine is seen to flow freely into the proctoscope.

RESULTS

Following the first stage, in which both ureters are imbedded simultaneously, the blood urea is usually increased for 3 or 4 days, corresponding to a temporary postoperative anuria. The animals drink and eat very little during this time. They then begin to drink and urinate freely, and the blood urea returns to normal in the course of the next 2 days. Some of the animals eat, drink and urinate normally the day following operation, and the blood urea is not increased during this postoperative period. None of the animals show any gross abnormalities. Intravenous urography reveals no dilatation of ureters or kidney pelves. When the second stage of the operation is done some 3 weeks later there is no evidence of hydroureters or hydronephrosis. The kidneys are macroscopically and microscopically normal.

The establishment of the communication between ureter and bowel at the second operation is accomplished with a minimum of healing surfaces. The blood supply to the healing mucosal edges is unimpaired, and so fibrosis must be minimal. Urine flows freely into the proctoscope as soon as the opening is made between the ureter and bowel. At no time has there been anuria during the second postoperative period. The urea content of the blood rises somewhat in the course of a few days and fluctuates from day to day, apparently depending upon the amount of water and nitrogenous material reabsorbed from the bowel. Intravenous urography again reveals normal outlines of kidney pelves and ureters.

SUMMARY

1. A new technique is described for ureteroenterostomy.

2. Complete surgical asepsis is maintained.

3. Healing between ureter and bowel in the absence of infection is insured.

4. A non-fibrous, patent communication with a check valve control is established between ureter and large bowel.

5. Postoperative dilatation of the urinary tract has not occurred.

The author wishes to acknowledge the able assistance and helpful suggestions of Dr. H. E. Shih, of the Peiping Union Medical College, during the latter part of this investigation.

EDITORIALS

SURGERY, GYNECOLOGY AND OBSTETRICS

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APRIL, 1935

INFECTIONS OF THE HAND

IN spite of the contributions and teachings of Kanavel in Chicago, of Auchincloss and his students in New York, of Bunnell in San Francisco, and of many other interested workers in many parts of the country, the toll of time, money and loss of function taken from the working man, in particular, as a result of infections of the hand is still far greater than it should be.

There are doubtless a number of factors responsible for this condition. One important one, obviously, is the lack of knowledge on the part of the public with reference to the proper treatment of open wounds. Too often the first thought that occurs to many an individual who has sustained an open wound of the fingers or hand is the confident assurance that has come to him over the radio only the day before, that if he will only be modern, and apply listovapopepsogine solution liberally to the open wound the outcome will be all that could be desired. Nor can the medical profession claim complete immunity to the siren wiles of the purveyors of merchandise. One need only recall the long list of "ideal" antiseptics and of healing ointments, now almost forgotten, to realize how credulous we have been in the past.

Another factor deserves still more thoughtful consideration. The care of infected hands is often relegated to the young and inexperienced surgeon. Particularly is this likely to be true in our large public institutions. The chiefs of the service cannot be bothered with the care of trivial conditions; they must center their attention on major surgery. The assistants take their cue from the chiefs; the contagion affects the residents in turn; and the patient eventually finds himself in the hands of the inexperienced junior interne. Too often not until some ill-advised incisions have been made, and not until the pathological process has advanced to such a degree that it is obviously a matter of grave concern are the older members of the staff called in, and the thoughtful attention that might have averted serious trouble if aroused earlier concentrated on controlling what has become a life-threatening infection, or on attempting to save the seriously injured structures so essential to normal function.

This condition reflects in turn an attitude which prevails in many medical schools. So much attention is devoted to thyroid surgery, surgery of the biliary tract, of the digestive tract, etc., and perhaps to the rare and dramatic surgical lesions that are engaging the attention of some member of the surgical department that little time is left for consideration of the more prosaic but surely much more important conditions resulting from wounds and infections. As a result, the student con-

fronted with such cases at the outset of his hospital career has no definite conception of the problem involved or how to meet it.

A member of the surgical department of one of our large medical schools was asked recently whose duty it was to consider with the students the problems of infection and injury. After a moment's thought he replied, "I don't believe there is anyone in our department who is particularly interested in minor surgery." As long as infections of the hand, and in fact infected wounds of any part of the body, are considered minor surgery we will not make much progress in the surgical care of patients with infection and injury.

It should hardly be necessary to repeat what every first year medical student knows, and what the senior student too often has completely forgotten, that the hand is a marvelous and intricate mechanism. When this delicate mechanism is entrusted to the care of the novice in surgery, armed with a sharp scalpel and an urge to "do things," the result is often unfortunate.

The remedy, I think, lies in our own hands. It involves:

1. Greater emphasis in our surgical teaching upon basic surgical principles, and more specifically upon accurate diagnosis and proper treatment in cases of infection and injury.

2. Proper emphasis on these problems in the hospital, and delegation of the responsibility for their solution to the most experienced men on the staff. It is interesting and stimulating to read a plea for organization of the surgical service of the hospital so that the patient with a postoperative pulmonary embolus can be operated upon promptly and given a chance for life.[1] But from the standpoint of our obligation to the public, and of accomplishing the greatest good for the greatest number, is it not imperative that the surgical service should be organized so that patients with infection and injury can receive prompt and truly skillful attention?

3. Education of the public to the fact that in the immediate treatment of injuries simple soap and water cleansing, and protection of the open wound against infection from without, with the aid of a sterile dressing, constitute the best possible form of first-aid treatment and one which is based on sound surgical principles. SUMNER L. KOCH.

[1] Lilienthal, H. Pulmonary embolectomy, the need of organization for prompt surgical intervention. J. of the Mt. Sinai Hosp., 1935, vol. 1, 234.

JEJUNOSTOMY FOR RESTORING ELECTROLYTES LOST IN PERSISTENT VOMITING

IN the average instance, postoperative vomiting is readily combated by the well known routine of the indwelling stomach tube and the maintenance of water balance by administrations in the veins, under the skin, and by rectum. The unusually prolonged and unyielding type of postoperative vomiting presents an entirely different problem. It is a question whether the surgical profession, broadly speaking, appreciates all of the factors concerned in a favorable or unfavorable response to treatment. In the work of the average surgeon, a year rarely elapses without his experiencing one or more instances of excessive and prolonged postoperative vomiting and encountering therefrom an unexplained fatal outcome.

The usual story is that vomiting becomes excessive about the fifth day and that in spite of gastric decompression by indwelling tube and maintenance of fluid balance by administration of saline and glucose solutions in the veins, the vomiting continues, thus resulting in a tremendous loss of gastric and intestinal juices. With this excessive vomiting the abdomen shows no distention (often remaining flat) and frequently reveals peristalsis in the lower coils. There are occasional bowel move-

ments. The surgeon, after futile search, can find no explanation of an obstructive character for the continued and excessive regurgitation. The patient continues to lose in circulatory strength, blood volume, and suddenly in a few hours goes down hill rapidly to a fatal outcome. Evidently in these cases there is some type of mechanical block, probably œdema near or at the duodenojejunal junction. With obstruction at this point, vomiting immediately entails a total loss of all the digestive juices. Experimental work has definitely demonstrated that life is impossible without pancreatic juices, that with persistent vomiting not only is the pancreatic secretion lost but also the electrolytes (sodium and chloride ion) from the duodenal and gastric juices, and in addition there is loss of the bile and other intestinal juices secreted by the mucous membranes. Some authorities contend that loss of the bile salts, which activate the lipases and aid in the emulsification of fats in the intestines, is more serious than the loss of sodium and chloride ion.

Rowntree has estimated that the secretion of digestive fluids averages from 5 to 7 liters daily. Thus in prolonged excessive vomiting not only is there a tremendous loss of water, but more important there is the loss of the electrolytes and the hormones of the pancreatic secretion without which life is impossible. Surgeons generally are familiar with the invariably fatal outcome of prolonged duodenal fistula. This of course entails loss of pancreatic fluid and electrolytes with the resulting acidosis of the uncompensated type. Prolonged excessive vomiting not only results in a loss of bile, pancreatic and intestinal juices, electrolytes and tremendous quantities of water, but finally winds up with definite alkalosis from loss of gastric acids. While it is generally conceded that death following this condition is due to dehydration it is more important for us to remember that dehydration in turn is specifically dependent on the loss of electrolytes together with certain qualities of pancreatic secretion.

Wilkie called our attention to the fact that death in high obstruction is due to loss of all digestive secretions by vomiting. Clinically Brockman has reported remarkable relief from obstructive types of vomiting by the administration of bile in the vomitus by rectum. In the past few years a great many surgeons have adopted the principle of returning vomited digestive fluids back to the patient by rectum yet we must remember that absorption by rectum as compared to the jejunum represents a ratio of about one to four. The principle, however, should be utilized early in every case of postoperative vomiting. White and Fender demonstrated experimentally that death in animals in simple high obstruction could be prevented by returning the vomited material into the small intestine below the point of obstruction.

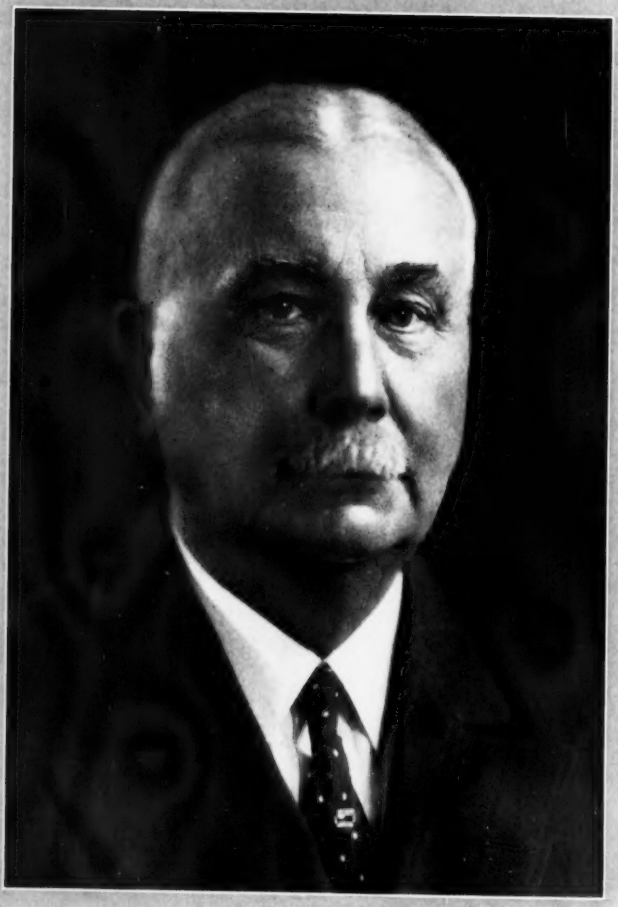
This very brief statement of relative facts concerning the fatal outcome in some cases of prolonged and excessive vomiting leads to the suggestion that death might be circumvented in many cases of duodenal fistulæ or unyielding excessive vomiting from simple duodenal obstruction by the institution, promptly, of a jejunostomy and the return of all vomited digestive juices into the jejunum. We have had the opportunity to observe the efficiency of this method on four occasions and consider that the maneuver of jejunostomy with return of all vomitus into the jejunum is definitely a life saving measure. The step is to do a jejunostomy and connect directly to the jejunostomy tube an indwelling nasal tube which has been passed into the stomach or duodenum. As a rule the drainage of the gastroduodenal juices readily run over through the stomach tube into the jejunal

tube. If there is vomiting with regurgitation around the indwelling stomach tube this should be immediately and totally returned, with aid of a funnel, through the jejunal tube. There has been no difficulty in the jejunum tolerating as much as one pint of vomitus returned at one time. The general influence on the patient for good is immediately noticeable. It is of course assumed that the electrolytes of sodium and chloride ion together with sufficient water will be administered by vein and under the skin. Often it happens that these routes have been more or less exhausted and the making of a jejunostomy with return of vomited digestive juices will become the determining factor between life and death. Especially consoling is the fact that the jejunostomy provides an avenue for administering water, hypertonic saline, and nourishment without further delay.

Recently a case of gangrenous pancreatitis showed every tendency toward recovery after surgical drainage until the tenth day when vomiting and excessive loss of digestive juices began and none of the usual routine measures would bring relief. Over the previous 10 days parenteral administration of saline solution, water, and glucose had supported the patient and this method was now becoming intolerable and insufficient. The question of intestinal obstruction anywhere below the duodenojejunal junction was ruled out and without further delay jejunostomy was promptly done and the indwelling gastroduodenal tube through the nose was immediately connected up with the jejunostomy tube. All drainage from the nasal tube and all regurgitant digestive juices around the nasal tube were immediately returned to the jejunum. Water, hypertonic saline, and gradually nourishment was given through the jejunostomy tube. The patient's improvement was almost immediate and continued to final recovery. Another case of duodenal and biliary fistula showed immediate favorable response to the return of all the fistulous discharge together with fluids and nourishment to the patient through a jejunal tube.

Surgeons should remember in every case of postoperative vomiting that the loss of digestive juices has a serious effect on blood volume through a sacrifice not only of water but important electrolytes representing inorganic factors in the composition of digestive secretion. Loss of electrolytes means dehydration and dehydration means reduction of the volume of blood plasma and increase of concentration of plasma protein. These physical changes together with increase of viscosity and a rise in the red count must interfere with the function of the blood as evidenced by huge reduction of the oxygen content of venous blood in duodenal obstruction and finally in the development of a lethal alkalosis as proved by the high carbon dioxide combining factor.

The moral is that prolonged and excessive vomiting is often, *per se*, fatal; that jejunostomy with gastric tube hook-up is simple and devoid of danger. Most important, in duodenal fistula or duodenal obstruction, jejunostomy should be done early for restoration of lost digestive fluids before lethal dehydration can take place. ROBERT LEE PAYNE.



L. L. McArthur

MEMOIRS

LEWIS LINN McARTHUR

IN the death of Dr. Lewis Linn McArthur the medical profession lost one of its most distinguished members and the surgical profession one of its leaders. Dr. McArthur possessed qualities of mind and heart that are not often to be found in the same degree in one person, and he had in unusual measure the confidence and respect of all who were associated with him and the love and appreciation of his friends. As a surgeon he never grew old, but was interested and interesting to the end.

During the many years I knew Dr. McArthur, I was impressed with his surgical acumen and his ability to adapt to the individual patient the surgical procedures to be carried out. He had a rare understanding of surgical pathology, and what might be called a flexibility of operative technic. I have seen him in the middle of an operation, on recognizing that the procedure he had planned was not best adapted to the case, change from one method to another, with complete success. He insisted that the tissues at operation be handled with extreme gentleness, and that meticulous care be exercised in all coaptations.

But more than technical skill, he had a humane understanding of the emotional states from which the patient suffered. He appreciated the natural desire of the patient not only to feel well but to look well, and he wanted to be of aid, in building up not only his physical but his mental condition. In his work he was a marked individualist. He took the most intense personal interest in each case, always visiting his surgical patients at least twice daily.

It would appear sometimes that the modern surgeon thinks of surgery only from the standpoint of the operation, the after-care of the patient, and the physical results. He carries out with the most scrupulous attention the necessary technical procedures, but not always does he possess that kindly spirit of helpfulness which is essential to carry the patient through an operation, to instill the confidence and courage which so often bring the patient safely through a crisis he might not otherwise withstand.

Perhaps this characteristic of spiritual understanding comes to those who in their earlier years were successful general practitioners, that is, practicing not only surgery but medicine as related to surgery. Perhaps the ability not only to acquire the confidence of the patient, but to deserve it, to see what the patient desires and

needs, comes through the sixth sense we call intuition, which in turn comes from wide experience and deep sympathy for and devotion to the patient, giving to the possessor remarkable ability to achieve results.

Dr. McArthur had a large surgical practice which took up his time to an extraordinary extent, and he was a successful surgeon because his skill and his humanity enabled him to carry many patients through most serious operations. He was a general surgeon to the last, but like all general surgeons of the older school, the pressure of patients afflicted with certain types of disease compelled him to restrict his work to rather definite fields.

Among Dr. McArthur's numerous contributions to surgical science, those in which he felt the greatest pride were: (1) the muscle-splitting incision for approach to the appendix, which is usually attributed to McBurney (among the papers found in his desk after his death was a letter dated 1894 from McBurney acknowledging Dr. McArthur's priority); (2) the autoplastic fascial repair of inguinal hernias, used in the McArthur clinic as standard since 1896 and recently popularized by Gallet; (3) the supra-orbital approach to the pituitary; (4) the use of the gall bladder or common duct for the administration of fluids; (5) the buried catheter method of common duct and ureteral repair; (6) the "tobacco pouch" method of nephropexy; and (7) the method of rectosigmoidostomy for complete rectal prolapse.

The many scientific papers which Dr. McArthur contributed to the various medical societies are a measure of his intense interest and activity in his profession. They are too well known to require discussion. Dr. McArthur was a member of his county and state medical associations in Illinois, of the Chicago Medical Society, of which he was a past president, of the Chicago Pathological Society, the Chicago Gynecological Society, the Institute of Medicine of Chicago, the International Surgical Society, the Colorado State Medical Association, the Western Surgical Association, the American Medical Association, the American College of Surgeons, the Society of Clinical Surgery, and the American Surgical Association, of which he was president in 1922.

Lewis Linn McArthur was born January 23, 1858 in Boston, Massachusetts, the son of Major Joseph Hunter McArthur, U. S. Army, and Julia Woodworth McArthur, daughter of Samuel Woodworth, poet, who perhaps was best known through his well-known poem "The Old Oaken Bucket."

In 1878 Lewis L. McArthur matriculated at Rush Medical College and was graduated in 1880. He won first place in the competitive examination for internship at the Cook County Hospital, where he served 2 years. On completion of his internship he spent 2 years in Vienna and Heidelberg in postgraduate study before returning to Chicago to take up his life work. He fitted out the first opsonic index laboratory in Chicago and sent his first assistant, the late Dr. John Hollister, to London for a year, about 1900, to study there the method of Wright.

In 1917, he received the commission of Major in the Medical Reserve Corps of the U. S. Army, and he organized and was director of U. S. Base Hospital No. 14. He was made Chevalier of the Order of Leopold of Belgium in recognition of his service to Belgium during the war.

Dr. McArthur is survived by three sons, Dr. Selim W., Lewis Linn, Jr., and Billings Meacher McArthur. Dr. Selim W. McArthur was fortunate in receiving his surgical training under his father. He has continued on the staff of the hospital where Dr. McArthur did much of his life work, and he well fulfills the tradition established by his father for skilled and humanitarian service. W. J. MAYO.

THE SURGEON'S LIBRARY

REVIEWS OF NEW BOOKS

VOLUME X of the *Cyclopedia of Medicine*[1] now appears and it covers the section *PRE* to *RHB*. The first subject covered is pregnancy, parturition and puerperium, and the first 20 pages are given to the anatomy of the female generative organs and the pelvis. The next 20 pages cover maternal care, so that a great amount of detail is given to each topic, the whole subject requiring 362 pages. The next section covers the anatomy, physiology, and disorders of the prostate and the section on treatment is filled with fine illustrations and drawings. A large section of 600 pages is given to the phases of radiology. "Remittent fever," "reticuloendotheliosis," and "rhubarb" indicate the great spread of topics discussed. The whole volume of 1,167 pages is well printed on good heavy paper and bound in a firm, green colored binding. Vols. X and XI conform nicely with the high caliber of the preceding volumes. Volume XI covers subjects occurring alphabetically between the letters *RIC* and *TEL*. Individual topics are contributed by men familiar with the subject so that the associate editors are found widely scattered. The first topic in Volume XI is that of "Rickets" written by Drs. Park, Eliot and Bromer. Rickettsia, rubella, saccharin, scarlet fever, silver, skin grafting, social service, spinal cord disorders, spleen, sprue, staining methods, stomach including its surgery, strophanthus, syphilis, ending with tar and tartar indicate the scope of medicine, surgery, pharmacology, and laboratory methods described. The volume consists of 1111 pages of well printed, well edited material in the conventional rigid binding.

The *Cyclopedia* is edited by Dr. George M. Piersol, and the review of the first seven volumes, which appeared in the December, 1933, issue of SURGERY, GYNECOLOGY AND OBSTETRICS (p. 822), covers the detail of the plan. M. H. BARKER.

STUDENTS and gynecologists alike will welcome this second edition of Curtis' *Textbook of Gynecology*.[2] While adhering to his original idea of having the book reflect his own viewpoint and practice, he has lengthened it by some 107 pages, thoroughly revised the illustrations, and increased its written material by about 70 per cent. This is a textbook and, therefore, covers, as we would expect, all of the frequently encountered gynecological conditions—the infections, the neoplasms, the injuries of childbirth and displacements, and the functional disturbances. Its great value lies in the fact that it is the work of a master gynecologist who presents the subjects of gynecology to us with remarkable clearness, and, considering the size of the volume, with surprising thoroughness. Everywhere he has given us the results of his own rich clinical experience and laboratory investigations. In 4 years there has been naturally a slight shifting of his viewpoint on several subjects, a thing that may be sensed by those who are familiar with both editions. The feature that is perhaps of greatest interest is the chapter on "Internal Secretions." The author has condensed into a chapter about all that is really known about this intricate subject, and it is exceedingly clear and well done. He has at various points in the book sought to couple up this knowledge with the treatment of various disorders. He remarks "despite the meager progress in treatment we are justified in assuming that further developments are imminent. Moreover, dependable tests for early pregnancy, for death of the fetus *in utero*, and for determination of the postoperative care of chorio-epithelioma merit inclusion in the list of advances made in the care of gynecological patients." RICHARD R. SMITH.

A FITTING monument to the work of Dr. Wilmer, who is retiring as chief of the Wilmer Institute of Johns Hopkins University, is found in *Atlas Fundus Oculi*.[3] The color plates of the fundus are by Annette Burgess, an artist trained by Dr. Wilmer especially for this work, and are made from his own patients and those of a few colleagues. They are beautifully reproduced, and represent a satisfactory variety of conditions seen with the ophthalmoscope. Only one criticism might be made of the illustrations as a whole—the light reflexes, the appearance of nerve fibers in the fundus and that of fine granular pigment appear in some cases to be exaggerated.

The careful description of the normal fundus and its common slight congenital abnormalities such as hyaloid remains on the disc is well done and of great practical value, since, as the author points out, it is common to see these mistaken for various conditions indicating disease. Thus the disc in high hyperopia

[1] THE CYCLOPEDIA OF MEDICINE. George Morris Piersol, B.S., M.D., Editor-in-Chief, and Edward L. Bortz, A.B., M.D., Assistant Editor. Vols. x and xi. Philadelphia: F. A. Davis Co., 1934.

[2] A TEXTBOOK OF GYNECOLOGY. By Arthur Hale Curtis, M.D., 2d ed. Philadelphia and London: W. B. Saunders Co., 1934. First edition reviewed 1933, 53: 118.

[3] ATLAS FUNDUS OCULI. By William Holland Wilmer, M.D., LL.D. ScD. Introduction by Warfield T. Longscope, M.D. New York The Macmillan Co., 1934.

is properly distinguished from that in early optic neuritis.

The findings in each case are briefly and simply described, with a minimum of discussion as to vexing questions of pathogenesis. Perhaps there is too little discussion of certain conditions. Thus "colloid excrescences" are described as such, and properly illustrated in Plate 86, yet it might have made the matter clearer to state that they are hyaline deposits on the lamina vitrea of the choroid and hence always very deep in the retina and seen through a certain of retinal tissue. His other illustration on Plate 84 of "colloid excrescences" seems to represent a somewhat different type of deposits which is located in the retina itself. The author's use of the term "druses," an Americanization of the German "Drusen," usually employed as synonymous for "colloid excrescences," is here noted by the reviewer for the first time.

The fundus conditions observed in hypertension, arteriosclerosis, nephritis, and diabetes are beautifully illustrated, and brief summaries of physical and laboratory findings in the illustrative cases are included. Enough distinction is not made between conditions due to arteriosclerosis and the type of change called by Fishberg "hypertensive neuroretinopathy" which distinction seems to be justified by recent additions to our knowledge of cardiovascular pathology. Especially valuable is the representation in several cases of the changes in individual lesions. A fine example is that illustrating the progress of a solitary tubercle of the choroid in Plates 39 to 41. The book will prove exceedingly useful to one studying ophthalmoscopy, as a reference book in any ophthalmologist's library and especially in institutions where ophthalmoscopy is being taught.

SANFORD R. GIFFORD.

FORMER editions of Jordan's well known student text[1] on histology have been reviewed in this journal. It is a useful student aid of the traditional type which furnishes reliable information on the more static features of tissue structure and organ architecture. Indeed, the chief suggestion toward improvement might well be the adoption of a dynamic viewpoint throughout.

Each successive edition has shown some improvement, although no radical departures have been made from the original plan, scope, treatment, or phraseology. The present (sixth) edition follows its predecessor closely except for a change in format. A larger page of more richly sized stock has been adopted, evidently to the end of improving the quality of the halftone reproductions. Slight gains for the better have been made in this direction. Any such advantage, however, is largely offset by the larger, thicker, and heavier volume which has resulted. A five pound book is a distinct burden to read, and this volume now outrates all American competitors both in bulk and weight.

Some pedagogical losses have been incurred in the resetting. One is the placing of all subordinate descriptive matter in full size print. Discussions of blood, lymphatic, and nerve supply, together with many lesser descriptive details, not only would appear to better advantage in fine print but also would help reduce the present cumbersome volume size. Toward the same end a still more drastic change could be made. This would consist in the total elimination of more than 100 pages devoted to a list of illustrations, a chapter on elementary histological technique, and a section on detailed laboratory directions designed to fit the author's own course but of no practical value to others. Any loss to the student would be negligible.

The greatest gain in the present edition has been the introduction of some 45 additional or replacing figures. Beyond this the preface is misleading as to the amount of new material presented. The Golgi apparatus, reticulo-endothelial system and neuroglia receive new treatments. The account of gametogenesis has been condensed. Otherwise the reviewer finds almost no changes or new entries of significance.

L. B. AREY.

THE second edition of Kuntz's well known treatise[2] on the autonomic nervous system will find a welcome in the libraries of many a neurologist. Professor Kuntz has brought up to date the only book of its kind written in English and has thus increased its value as a reference work for those interested in the visceral side of the nervous system. He has rendered a distinct service in furnishing synopses of nearly all the important original investigations in this field and in supplying a magnificent bibliography.

All parts of the book will not share like praise by various readers. Some will feel that such sections as that on the development of the sympathetic system or that on the innervation of skeletal muscle are not unbiased by the author's own studies. Two criticisms of a general sort which are applicable to the book as a whole might be considered. It reads in monotone even to one intensely interested in the subject, a point which quickly discourages students from making as extensive use of the book as they should. One of the reasons for this fault may appear in another; the author fails to deal with subjects as critically as he might. He has not evaluated results of original investigations as carefully as should be done to give life to the book.

WILLIAM F. WINDLE.

THE second edition of Bell's *Pathology*[3] contains 140 more pages than the first edition, the various topics are discussed more fully, a large amount of new material has been introduced and an entire new chapter on diseases of bones and joints has

[1] A TEXTBOOK OF HISTOLOGY. By Harvey Ernest Jordan, A.M., Ph.D. 6th ed. New York and London: D. Appleton-Century Co., Inc., 1934.

[2] THE AUTONOMIC NERVOUS SYSTEM. By Albert Kuntz, Ph.D., M.D., 2d ed., rev. and enl. Philadelphia: Lea & Febiger, 1934.

[3] A TEXTBOOK OF PATHOLOGY. Edited by E. T. Bell, M.D. 2d ed. Philadelphia: Lea & Febiger, 1934.

been added. But the volume is still more than two hundred pages shorter than its nearest competitor. This is not necessarily a defect, for the book is well written with a commendable economy of words. Any contribution to the subject from the Department of Pathology of the University of Minnesota will command attention, and the present volume is no exception. It is a work of collaboration by E. T. Bell, B. J. Clawson, Hal Downey, J. S. McCartney, and C. J. Watson.

Dr. Bell contributes all of the chapters on general pathology except that on "The Mycoses or Fungus Infections." This latter was written by J. S. McCartney and is praiseworthy because of its completeness since it includes several diseases omitted in most of the textbooks of pathology. The chapters on "Diseases of the Urinary System" by Bell, on "Diseases of the Heart" by Clawson, and on "Diseases of the Blood" by Downey, are essentially short monographs on these subjects and fulfill expectations based upon the reputation and experience of each of these authors in the field in which he writes. These are probably the most notable chapters in the volume and are beautifully illustrated.

Many subjects are presented with unsatisfying brevity. But few, if any, significant facts are omitted, although they may not be fully explained. The junior and senior medical student will be pleased to find listed in the index and at least defined in the text many rare diseases with which his clinical teachers delight to puzzle him, such as Milroy's, Landau's and Kienboeck's diseases. This fact, among many others, justifies the statement of the editor in the preface that the authors "have attempted to present the facts of pathology in such a way that the student will look upon clinical medicine as a direct continuation of his pathological studies and not as an abrupt entrance into a new field."

The surgeon will be interested, among other things, in the discussion of "the value of the biopsy and how it should be performed," at the end of the chapter on tumors which was written by Bell. After a brief discussion of the prejudice of many physicians against biopsies, Bell concludes: "If we discard the biopsy it will mean removal of legs for giant cell tumors, radical breast operations for fibro-adenoma and cystic disease, removal of jaws for benign ulcers, etc. Surely the theoretical dangers of a biopsy do not offset its known value." Bell is not impressed with the alleged advantages of grading tumors as it is usually done, on the basis of "the relative number of differentiated and undifferentiated cells." He believes that "the malignancy of a tumor should be judged by its most malignant part and not by its average structure."

J. P. SIMONDS.

THE textbook of Cowdry[1] brings the freshest breath to the field of histology that has been experienced for some years. Here is a novel attempt by one of America's foremost cytologists to write dynamic histology, to interpret structure through function, and to present a digest of the best contemporary thought. Even the table of contents reflects the physiological approach, as witness the main section heads: (1) water, the essential vital medium; (2) the blood, the principal integrator; (3) absorptive drainage into blood; (4) chemical integration by endocrine products in blood stream; (5) intake of water, nutriment, accessory food factors and removal of waste; (6) oxygen consumption and carbon dioxide elimination; (7) regulation of constitution of blood and removal of waste; (8) rapid neural integration in response to internal and external stimuli; (9) architectural support; (10) perpetuation of the race; (11) unification, protection, and adjustment. From these headings the plan of organization can readily be grasped, and it will be immediately manifest how radically the traditional arrangement has been altered. Furthermore, there is no account of the cell as such; its various features are discussed disconnectedly wherever they are best illustrated by the subject matter at hand. Even the tissues experience somewhat the same fate. Epithelium is first described under the thyroid gland, while the connective tissues and muscle are discussed in almost the last chapters.

From this superficial recounting of certain obvious peculiarities, it is plain that Professor Cowdry's text is indeed something out of the ordinary. When next the subject matter is scrutinized it is equally clear that no attempt has been made to present a well rounded whole. Tendons occupy eight lines and cartilage less than a page; the ureter gets no specific description, but its innervation is fully discussed; hair, sebaceous glands and sweat glands receive but one and one-half pages, whereas the pituitary gland claims nineteen; the muscles are limited to ten pages, while the blood cells alone take sixty-two.

Any criticism of these departures from the ordinary treatment is partly disarmed at the outset by the preface, from which pertinent quotations can be made to advantage.

> The time is passed when a broad, even presentation of the microscopic structure of the whole body is needed. Much that is important must be omitted in order to present the most vital subjects at all adequately. Justification for this is found by comparing standard works on physiology and histology. In the latter many pages are devoted to structures which the physiologist does not deem worthy even of mention. Concentration on a few subjects affords opportunity for the presentation of some of them in their proper setting, for emphasizing the value of experimentation and of what constitutes evidence. . . . What to omit and what to stress is the question. . . . The usual plan is to begin with a description of a typical animal cell, then to pass to the various tissues and finally to the several organs. This is illogical and time-consuming. . . . These [cells and tissues], likewise, do not exist for themselves and by themselves, but function only in so far that they are integrated with the rest. Consequently an attempt is made to build up, as the discussion advances, a conception of cells and elementary tissues in the many environments in which they normally exist. Organs and systems are stressed in order to fulfill the principal aim of the volume, which is to relate structure and function in the whole body—itself the real physiological unit. . . . A just criticism of most textbooks of histology is that they repeatedly illustrate the

[1] A TEXTBOOK OF HISTOLOGY; FUNCTIONAL SIGNIFICANCE OF CELLS AND INTERCELLULAR SUBSTANCES. By E. V. Cowdry. Philadelphia: Lea & Febiger, 1935.

obvious. Figures consuming much space merely show at a glance what the actual preparations will lead the students to discover for themselves—a much more healthy experience. They are beyond the picture book stage. . . . As indicated in the list of contents, the central theme of the book is the blood vascular system—the great integrator. . . . The goal in view is that the students shall gradually visualize more and more accurately the wonderful reactivity of the minute structure of the human body in terms of biochemistry, physiology, and pathology and, finally, in practice shall institute measures of assistance with due caution. . . . Controversial subjects have in no way been avoided, rather have they been sought out and aired. An effort has been made to present the evidence fairly and to acknowledge the labors of leading investigators.

Doubtless some of these viewpoints will not find favor with many teachers; others while sympathizing with their spirit will consider them impractical in application. The reviewer is convinced that our beginning medical students, coming as they do without any definite understanding of even the nature and properties of protoplasm or of the organization of a cell, must learn to creep before they can be expected to walk—to say nothing of running. For this reason he holds that since structure is the primary concept to be gained (before function is made to vitalize such information), a presentation which does not become wholly subservient to an ideal functional continuum is the simplest and wisest to pursue. Otherwise, confusion and discouragement to the beginner is both predictable and certain. On the other hand, with a moderate amount of previous histological experience, study of this book should be a thrilling adventure. The greatest value of Professor Cowdry's text will perhaps be as a book in which collateral reading is expected. It would make, for example, an admirable companion volume to the Maximow-Bloom text which is easily the foremost in the orthodox field. With such guides the student will be enabled to get an insight into histology such as has not been possible before through the medium of the printed page.

To the medical practitioner Cowdry's *Histology* can not be too strongly recommended. It is no ordinary compilation, but rather a mine of authoritative, up-to-the minute information and interpretation. If the clinician can find a page which does not offer valuable new viewpoints and unexpected rewards, then that man has been a prodigious reader and analyst of current histological literature and can well-nigh qualify as an expert in the subject.

L. B. Arey.

FOR the preparation of his book,[1] Meaker set out to assemble completely the knowledge now well established concerning involuntary sterility, discounting the traditional fallacies and including all the modern discoveries. The purpose was accomplished admirably but this was to be expected because Meaker is one of the foremost authorities on the subject of sterility. The book is divided into three parts. In the first part the author takes up the causes of human infertility and he discusses them under nine headings. The second part is devoted to the question of the diagnostic study of the sterile mating. Not only are the gynecologic aspects discussed but also the urologic ones. There are excellent chapters on the study of the male genitalia and semen. There are also lucid descriptions and instructive illustrations of transuterine insufflation, hysterosalpingography, and postcoital examinations. Three chapters are devoted to endocrinologic investigations.

Meaker points out the value of group study of cases of sterility. In 1927 he formed a clinical group with a urologist, an internist, and an endocrinologist in order to study sterility cases more thoroughly. The third part of the book deals with the treatment of sterility and in this section, Meaker points out the great value of a clinical group for the intensive study of sterility cases. Before he formed the clinical group the incidence of pregnancy in his sterility cases was 24.6 per cent whereas the frequency of gestation has been 50 per cent in the series of cases studied by the group. Meaker realizes, however, that for many reasons, groups are not feasible in all cities, hence he describes the minimum treatment which a physician can carry out by himself. Urologic as well as gynecologic treatment is outlined. As regards surgery to overcome tubal occlusion, Meaker has had phenomenal success, because of the 19 patients on whom he performed salpingostomy, 9 conceived. Concerning endocrine causes of sterility the author found the prognosis to be fair in the pituitary cases, excellent in cases of thyroid deficiency and poor in failure of the ovaries. Contrary to most physicians, Meaker has seen good results follow the oral use of anterior pituitary preparations.

Throughout the book there is evidence of the author's comprehensive grasp of the subject of sterility, not only of the physical factors involved but also of the psychic ones. Excellent advice is therefore given concerning both factors. The book deals with its subject so completely and rationally that it should be carefully studied not only by physicians who are especially interested in sterility, namely, gynecologists, urologists, and endocrinologists, but also and more particularly, by general practitioners.

J. P. Greenhill.

THIS imposing volume[2] by Brailsford, dedicated to the memory of Sir Robert Jones, constitutes a distinctly important event in the evolution of the literature of clinical radiology. Beautifully printed on excellent paper, in large type with well spaced lines, its excellent diction literally tempts the reader to linger longer in the study of its rich pages. Chapters are devoted to the normal findings in the skeleton at birth and the various congenital, dystrophic, and traumatic factors influencing the rate of ossification and the aberrations from the normal. After

[1] Human Sterility; Causation, Diagnosis and Treatment; A Practical Manual of Clinical Procedure. By Samuel Raynor Meaker, M.D. Baltimore: The Williams & Wilkins Co., 1934.

[2] The Radiology of Bones and Joints. By James F. Brailsford, M.D., M.R.C.S. (Eng.). Baltimore: William Wood & Co., 1934.

a few introductory pages of general discussion of the various pathological findings, the major portion of the book is devoted to a detailed study of the various regions of the skeleton and the lesions affecting them. The last hundred pages are devoted to a general consideration on bone changes in systemic and localized disease, and a final chapter on bone tumors. A lengthy bibliography concludes the work.

Special stress is laid upon the value of periodic radiographic examinations in studying normal and irregular growth, in observation of the progress of systemic and localized disease, and before, during, and after the exhibition of various forms of treatment. The lapse of a few weeks may supply the clinician with X-ray evidence of great value, though the preliminary X-ray findings may have been of slight or no value. Apart from setting forth concisely an account of the bone changes seen in health and disease, the author's aim has been to indicate the significance of the roentgen findings. Every effort has been made to include in this work the most recent advances already recognized in roentgen ray departments and journals but not yet adequately appreciated or accepted by general textbooks. In the opinion of the reviewer, the task has been well done. JAMES T. CASE.

IN the work entitled *Tuberculosis of the Lymphatic System*[1] the author covers the subject in great detail with particular reference to the more common regions of gland involvement. His introductory chapter, wherein he traces tuberculosis of the glands back to the early Greek and Egyptian conceptions of the etiology, is particularly interesting to the student of medical history.

The author discusses in great detail bacteriology, pathology, chemistry, and pathogenesis in this condition. The anatomy of the lymphatics is extensively described and beautifully illustrated. In the two chapters devoted to the subdivision of cervical lymph nodes, a clear picture is given of their involvement, the pathology, clinical course, diagnosis, and differential diagnosis, and the treatment, medical and surgical.

Of great practical interest is the discussion of tuberculosis of the tracheobronchial and abdominal lymph nodes. The outline of the care of the disease is a résumé of the treatment, in all its phases, and is especially to be commended for the statistical details of medical and surgical results.

This book can be recommended as an excellent text for the student and the general practitioner. The index, which is placed at the conclusion of each chapter, is an elaborate and very useful compilation of all recent writings on the subject.

W. A. HENDRICKS.

AN *Introduction to Gynecology*[2] is exactly what its name implies. This book was written primarily for third year medical students and is an outgrowth of a series of notes which the author has used for some time. The book is well written, each subject being presented simply, clearly, and briefly. It will doubtless prove to be a profitable and popular one for students.

This second edition has been made a more complete text by the addition of a short synopsis on treatment in each of the chapters dealing with diseases, although anatomy, pathology, and diagnosis are still emphasized. The bibliographies at the end of each chapter have also been expanded.

Recent advances in gynecology, such as the Friedman test for pregnancy, the Schiller test for carcinoma, and the use of the colposcope have all been included. It is unfortunate that the chapter on the endocrines has not been brought up to date in a similar manner. RALPH A. REIS.

[1] TUBERCULOSIS OF THE LYMPHATIC SYSTEM. Richard H. Miller, M.D., F.A.C.S. New York: The Macmillan Co., 1934.

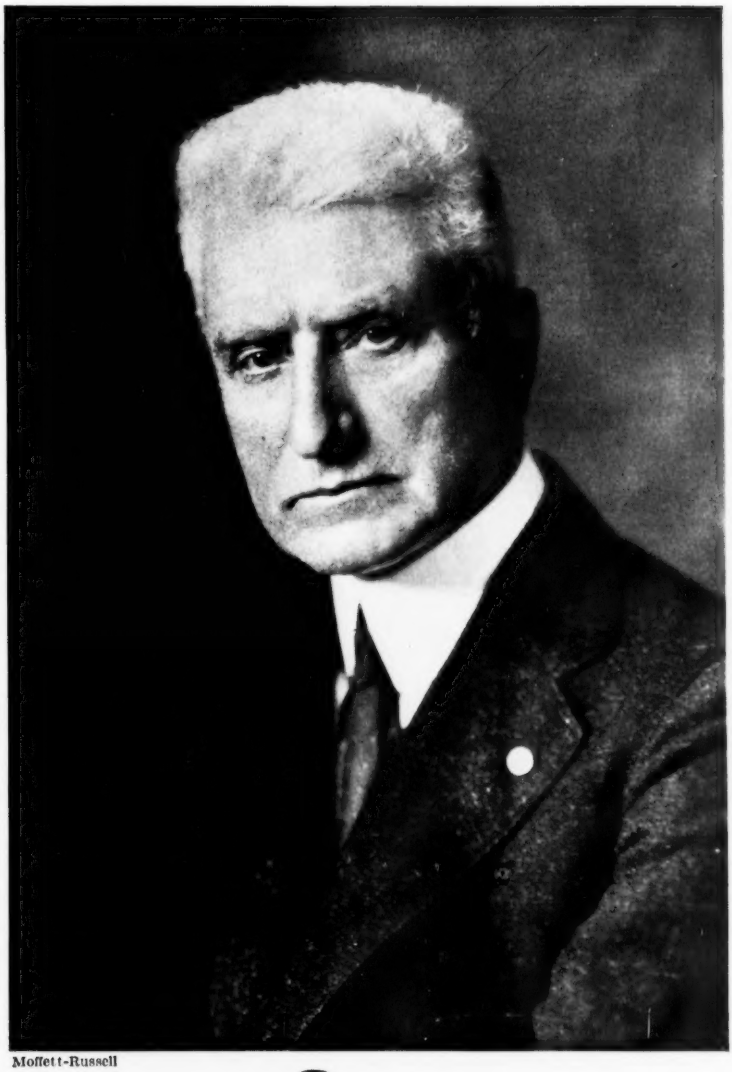
[2] AN INTRODUCTION TO GYNECOLOGY. By C. Jeff Miller, M.D. 2d ed. St. Louis: The C. V. Mosby Co., 1934.

In Memoriam

FRANKLIN H. MARTIN, M.D., F.A.C.S.

BORN JULY 13, 1857
DIED MARCH 7, 1935

FOUNDER AND MANAGING EDITOR OF
SURGERY, GYNECOLOGY AND OBSTETRICS
1905–1935



Moffett-Russell

Franklin H. Martin

FRANKLIN H. MARTIN

OUT of the hardy pioneer spirit of two families, the Martins of Canada and the Carlins of Pennsylvania, each coming west by caravans in the late 1840's and settling on adjacent farms in Wisconsin, grew the friendship and marriage of Edmond Martin and Josephine Carlin, to whom a son, Franklin, was born in 1857.

Franklin Martin's childhood was a happy one, in spite of the sacrifices of his pioneer forebears, the loss of his father in the Civil War, and the remarriage of his mother and the uniting of two families of children, the Martins and the Mungers. Under moral and religious influences, Franklin developed into a ruddy, sturdy, industrious lad, receiving his elementary education by the sweat of his brow.

Aunt Mary and Uncle Addison Carlin were a dominating influence for good in his life. He worked as farm hand, brickmaker, carpenter, janitor, and, later, as school teacher. In 1872 he left home for Minneapolis to study. In 1874 he returned to Wisconsin and entered Elroy Seminary. And then he decided to become a doctor.

His first professional hero was Dr. McLaren Miller, of Oconomowoc, and, later, Dr. William Spalding, of Watertown, in whose office he first worked as janitor, in his spare moments reading a book on anatomy, which he memorized.

In 1877 he came to Chicago with just enough money for carfare and tuition, and at the suggestion of Nicholas Senn, who signed his certificate of matriculation, he entered the Chicago Medical College, which is now the medical department of Northwestern University. It was here that he met as instructors Nathan S. Davis, Sr., William E. Quine, William Byford, Ralph Isham, John H. Hollister, and Edmund Andrews, all of whom were a great influence in his medical career. He loved the fire and enthusiasm of Quine, Davis, and Andrews, but he also loved the rockbound stability of John Hamilcar Hollister, who later became his father-in-law and who was a stabilizing and stimulating influence throughout his entire medical life.

His first domicile as a bachelor with young Frederick Parkhurst was at 1133 South State Street, where they cooked their own meals. During vacations of these years he returned to Wisconsin and again worked as brickmaker, schoolmaster, and carpenter, earning his own tuition and living expenses.

His first meeting with John B. Murphy and Lewis Linn McArthur was at the time they were taking Cook County Hospital competitive examinations.

He entered Mercy Hospital, where he worked with Dr. Lorenzo Potter as an interne, in 1880. He received his degree March 30, 1880, at the memorable old Plymouth Church at 26th Street and Michigan Avenue, which was to remain his church home until its closing in 1915.

During these medical school days he was given the opportunity to earn some money by caring for the patients of some of his professors. It was during these years at Mercy Hospital that antiseptic surgery and the bacteriologic theory of medicine began to take great hold and he was intensely interested in the first local attempts at the prevention of infected wounds and in the struggle against the spread of typhoid fever, which was at that time a serious menace to the health of Chicago.

In 1881 he began his medical practice, boarding with the Lord family at 2227 Wabash Avenue and meeting there his life long friends, Frank Bowles, James Chapman, and Charles Nicola. He joined Plymouth Church and met Isabelle Hollister, whom he courted, and married May 27, 1886. To her his autobiography is dedicated and with her he had a long and beautiful companionship. She it was who became the great inspiration of his remarkable career.

WILLIAM R. CUBBINS.

FRANKLIN H. MARTIN—THE SURGEON

FRANKLIN H. MARTIN graduated in 1880 from the Chicago Medical College which later became the Northwestern University Medical School. He served as an interne at Mercy Hospital, Chicago, during that period in the evolution of surgical technique when little was known of antiseptic surgery and practically nothing was understood of aseptic surgery. At this hospital, he received his first practical experience and training in the art of surgery under the tutelage of Edmund Andrews and other masters of surgery of that day. The death rate following major operations was extremely high in this era. The progress in knowledge and his own application of what it taught soon extricated Martin from the paraphernalia of antiseptic technique and he among the first in America began to practice aseptic surgery.

One of Dr. Martin's first contributions to surgery was the founding of a Post Graduate School and Charity Hospital and this accomplishment was predicated upon his understanding of the need for disseminating the rapidly increasing knowledge of aseptic surgery. He recognized that this information must be imparted to the older men in the field who were attempting to carry out surgical procedures in the outlying communities distant from teaching centers. This early thoughtful consideration of the need for the education of the actual workers in the field and his desire to help them was the motivating factor of his later activity in developing organizations for the elevation of the standards of surgery and the continuous education of all surgeons.

During the past twenty years, the honors and distinctions he has received as an organizer of the medical forces during the World War and as the instigator and the builder of the American College of Surgeons have led many to forget him as a surgeon. He was, however, always pre-eminently the surgeon and as such was held in high esteem by the men of the generation with whom he was associated.

In his early work as a gynecologist, before the perfection of aseptic technique made abdominal section safe, he did much to popularize the operation of tying off the uterine arteries through a vaginal incision, thus controlling the hæmorrhage and inhibiting the growth of myofibromata. As a result of this operation, otherwise hopeless cases were benefited and some cured.

Dr. Martin was a pioneer in experimental surgery on animals. His surgical curiosity led him to turn to an investigation of methods for perfecting a technique for the implantation of the ureters into the colon. The recorded data

of these investigations and the principles he established remain the foundation of all subsequent progress that has been made in this vexing problem. He performed one of the first operations for the removal of a diseased urinary bladder followed by the implantation of the ureters.

Surgical literature abounds with his contributions to a wide variety of subjects. These studies were made largely on conditions pertaining to surgical gynecology.

Dr. Martin's surgical acumen was great, and he frequently astonished his young associates by his logical and accurate diagnoses. He was averse to exploratory incisions for information and he never knowingly performed unnecessary or questionable operations. Frequently younger co-workers, who in their enthusiasm suggested operations not clearly indicated, were admonished to be conservative.

While at first the work he did was confined to gynecology, he became one of the outstanding abdominal surgeons of the West. His technique was most meticulous, his handling of tissues gentle and dexterous; though a fearless and rapid operator, he was never over radical, and his conclusions relative to pathological conditions and their operability were based on sound surgical judgment.

In that era when abdominal tumors were not recognized or attacked at an early stage of their growth, many patients came to his Post Graduate Clinic with enormous neoplasms. If the indication for operation was clear, with no serious contra-indications, he never refused to give these sufferers relief. A less courageous surgeon would have hesitated. The many excellent results in these seemingly hopeless cases attest the excellence of his judgment and the perfection of his technique. He was ever conscious of the human equation in all of his work.

He was a teacher of surgery from the beginning of his career. Any knowledge he imparted to his classes was founded upon wide reading and upon his personal and practical experience and observation. His clarity of thought and directness of expression made any subject under discussion readily understandable and his deductions were so logical and the conclusions so sound that they profoundly impressed his listeners. His commanding personality and dynamic force enhanced his ability as a teacher and these so impressed his assistants and younger associates that he passed on to them many of his attributes. As a developer of young men during their formative period in surgery he had few equals among the teachers in this department of medicine. All of the men who were his assistants and closest associates, many of whom later achieved success in their professions, were stimulated by his inquiring mind and influenced by his kindliness and generous consideration.

It was in the capacity of a teacher of surgery that he realized the necessity for a broader dissemination of the rapidly accumulating facts relative to practi-

cal surgery. This realization of the requirements of the men in the smaller communities who were doing surgery prompted the initiation and development of the Clinical Congress of Surgeons of North America.

His conception was that the surgeons who had fewer opportunities for study could learn faster and their knowledge of the basic principles of practical surgery would become greater if the chance were afforded them to watch the teachers of surgery and surgeons who had a larger surgical experience at work in their own workshops. Thousands of surgeons in the United States and Canada can bear witness to the value of his inspirational thought which originated in the mind of this Master Surgeon.

In later years came his dream of the American College of Surgeons, which he made a reality. The early vicissitudes and obstacles that were encountered in the organization of this great group of men did not daunt his untiring spirit; he recognized no barrier as insurmountable in achieving his purpose. As a result of his great vision, his courage, his indomitable will to succeed, and his unusual ability as an organizer, he has builded the American College of Surgeons which will endure in perpetuity as a monument to his understanding of the need for continous surgical teaching in its broadest sense.

Looking back over his completed life, we are impressed with his great vision, his love for his fellow man, his appreciation of the ways to advance human welfare through education, and his untiring energy and industry. His work, as he had outlined and planned it, was finished. His memory commands our respect and admiration for the manner in which he worked to the end with the same aggressive spirit and determination which characterized his long and useful life.

We honor his memory as one of the great surgeons of his generation.

FREDERIC A. BESLEY.

FRANKLIN H. MARTIN—THE FRIEND

A faithful friend is a strong defence: and he that hath found such an one hath found a treasure. Nothing doth countervail a faithful friend, and his excellency is invaluable.—Ecclesiasticus VI, 14 and 15.

THE passing of Franklin Martin removes a unique personality from the medical world. His versatility and the variety of fields in which he labored with such phenomenal success will naturally furnish ample material for an interesting chapter in American Medicine. Many will speak of him as an educator, an investigator, as an organizer and leader of men, but those who enjoyed the privilege of intimacy with him will think first of him as a friend.

It was a characteristic of his early boyhood to attract and attach to him his companions, as with a band of steel, and this power lasted throughout the years of a long and useful life. That the record may be clear, it may not be out of place to recall to the generation of physicians who have known Franklin Martin that he was one of those constructive or creative dreamers whose accomplishments are to be measured far more by what the man himself meant to others than by recital of achievements. From the dreams of this man, whom I have known intimately since 1900, have sprung the greatest surgical journal of the age—SURGERY, GYNECOLOGY AND OBSTETRICS,—the Clinical Congress of Surgeons of North America, the American College of Surgeons, and the Gorgas Memorial Institute.

How did this come about? I am powerless to suggest an answer other than may be implicit in the personality of one who was a friend and a man. When I say that he attracted friends, the wrong impression may be given, for it was not easy to win one's way into the inner sanctuary. He was by nature sensitive and shy, his approach quiet and dignified, no rush of insincere greeting to all and sundry. But, as one came in contact with this eminently sane and simple man, one became aware of his abundant resources, and perhaps suddenly a sly touch of his quiet humor would sweep aside the veil of shyness and let one glimpse the lovable being within. Certain elements we all instinctively recognize as essential in the true friend, among them, first, courage and kindliness and perfect candor. Courage was one of his chief attributes. He no doubt believed with Barrie, that "It is the lovely virtue—a rib of Himself that God sent down to His children." To those who knew him intimately it is needless to speak of his kindliness, his interest in the younger generation, and the promptness with which he recognized and encouraged merit in the younger members of the profession.

He was born on a farm and grew up among surroundings conducive to quiet thought. Much of his constructive thinking was done while walking in the great out of doors, for which his autobiography reveals his wholesome love. Nothing pleased him more than to have one or two of his friends join him in long walks, during which he discussed various problems in which he was interested. More than once flashes had come during these happy jaunts that later became basic principles in one of his projects. In his autobiography he mentions the Clinical Congress of Surgeons of North America as having been conceived as he walked the deck of a ship cruising in the Mediterranean, and those who have shared such jaunts with him can easily visualize his methods of thought and how his schemes of successful organization were launched. His love for out door life naturally attracted him to golf. His congenial side was at its best with his friends on the golf links, and for many years it was his chief recreation. Throughout this country and abroad there are many who remember and appreciate his charm, and a lasting friendship that started on the golf course.

He had a way of attracting and binding people to him in spite of the fact that he was often very frank in discussion. Fearlessness was one of his chief attractions, and even in hot argument he could maintain his point without losing his friends. There were necessarily many trying situations that arose during the development and direction of large organizations, but his keen insight, resourcefulness, judgment, and sincerity were never questioned, and his policies usually prevailed. When organization was finally effected, his friends remained his friends, for his candor and patient courage withstood the storm and stress of the process through which the end had been attained. After all, the true estimate of an individual can be best obtained from those with whom he labors daily. His subordinates adored him, labored constantly in his behalf, and respected his strict discipline, knowing full well that he observed equally with them the rules governing the organization.

No man liveth to himself alone, and it would be wholly ineffectual to offer such comments as I can make upon Franklin Martin without acknowledging in full measure the help he had from the very remarkable woman, his wife, Isabelle Hollister Martin. How well they understood each other, how much he deferred to her judgment; how wise she was in counsel, and how prompt he was to abandon certain problems of which she did not approve. Very few who did not know them intimately could appreciate how much she figured in his life, how necessary she was to his happiness, and how much his success has been due to her sound judgment, loyalty, and companionship.

Franklin Martin has left a splendid heritage to the medical profession. No bequest could be more useful than the sound principles he enunciated and wove into the fabric of the institutions he evolved for the benefit of the human race. To me the greatest inheritance is *The Joy of Living*, an *Autobiography*, which

typifies the man and the friend—for it expresses his attitude toward life. If one has read this fascinating book one will find how beautifully it expresses his ideas, and it shows, too, the real worth of the man and his confidence in his eventual success. From the very start of his life there is no note of appeal for sympathy for the hardships he endured, no appeal for pity, but through the narrative, as through his daily life, running like a thread of gold woven into it, there was *real* joy of living. And because of his simplicity, goodness, honesty, and sincerity, I know of no man in civil life who could, by the mere suggestion of needing or wanting them, summon more true and influential friends to his side.

The loyalty of his thousands of friends should be a pledge to foster and bring to full fruition the ideals and aims to which he devoted his life.

C. JEFF MILLER.

FRANKLIN H. MARTIN AND SURGERY, GYNECOLOGY AND OBSTETRICS

DR. MARTIN was fundamentally an idealist. A close personal and professional association for thirty-five years gives me some authority to speak. Few members of our profession have been more misunderstood. This misunderstanding arose because he was possessed of an extreme sensitiveness and a reluctance to approach people or speak in public that to many will seem unbelievable. As a result of this characteristic he often seemed autocratic, though this was a pure defense reaction. His own unintentional disclosure of this trait is found in a sentence taken from his autobiography, *The Joy of Living*. In speaking of his hesitation in beginning his first laparotomy he said, "Was I a coward? This was a bracing thought: 'Have the courage to do the thing you believe to be right but that you are afraid to do.' It became my slogan—a sentiment that during all my life has urged me to action, a few times (I hope) with success, and many times (I am sure) to my detriment."

His mind arrived with lightning speed at logical conclusions that most of us reached only after prolonged deliberation. His impatience drove him forward without the help of the support that would have come had he been content to wait until more slowly reasoning minds had caught up with him. This impatience helped to give the impression that his actions were often autocratic.

He had little or no desire to accumulate wealth. Money meant nothing to him but the means of establishing the children of his imagination. He realized, however, that for them to succeed and to attain permanence they must be placed upon a secure financial foundation. This is well illustrated by an account of the origin of SURGERY, GYNECOLOGY AND OBSTETRICS and of his plans for its ultimate disposition.

In December, 1904, Dr. Martin invited Drs. F. A. Besley, William R. Cubbins, John Hollister, and myself to his home at 3210 Lake Park Avenue, where he outlined his plan for the founding of the journal. His conception was of a journal divorced from any commercial association which should be founded, owned, and directed by members of the profession. It was to be an example of the best art of printing; it should present in adequate form the new developments of surgical science, but should always be a practical journal for practicing surgeons. It should be based on sound financial principles, yet never seek to pay dividends to stockholders; it should rather use any profits to increase the service of the journal and insure its stability.

Thirteen Chicago surgeons supported these altruistic principles by subscribing for small blocks of stock. Later when Dr. and Mrs. Martin began to plan for the disposition of the journal they purchased this stock at a profit to the original stockholders.

During thirty years under Dr. Martin's direction the journal has adhered to this original conception. From the beginning, Mrs. Martin has held a large part of the stock and she also has been in full accord with Dr. Martin's ideals. Because of Dr. Martin's wise planning the journal now rests upon a secure financial foundation, and owns the land and buildings at the corner of Rush and Erie Streets which adjoin the College of Surgeons.

Dr. Martin's manner of administering the various organizations for which he was responsible may be illustrated by relating his conversation with me when organizing the journal. He called me to his office and said, "I want you to assume charge of the scientific pages of the journal. Make the journal the best in the world scientifically and artistically. Do not be influenced by anybody or anything except the quality of the contributions and the limitations of space. That's all." While he always took a leading part in any expansion or new activity, yet never in the thirty years did he let personal friendship, expediency, or advertisers betray him into seeking to influence the acceptance of any article. Nor did he interfere in any way with the administration of the department. It was the same with other associates. He expected results but gave complete freedom of action and support in any difficulty. His loyalty to his associates and friends was so great that not infrequently it surpassed what should have been expected. Except in the face of incontrovertible evidence of incompetency, he defended them both privately and before the public.

The first number of SURGERY, GYNECOLOGY AND OBSTETRICS appeared on July 1, 1905, with the leading article by the chief of the Editorial Board, Dr. Nicholas Senn, upon "Iodine in Surgery, with Special Reference to its Use as an Antiseptic." The ideals of its founder and the character of the journal soon brought contributions from leading surgeons of the United States and Canada and from abroad in sufficient number to insure the journal's immediate success.

In 1908 a British edition was established under the editorial guidance of A. W. Mayo-Robson (later Sir Arthur) and other leading British surgeons. Five years later, after much thought, the INTERNATIONAL ABSTRACT OF SURGERY was added. It was determined that the ABSTRACT should be not a mere desultory collection of material but a discriminating selection from the world's literature by a board of specialists. Arrangements were made with the leading abstract journals abroad for the early exchange of their material. As a result a complete bibliography of the world's surgical literature has become a part of the ABSTRACT.

After Senn's death, Dr. John B. Murphy, whose enthusiastic support had been no small factor in establishing the journal, became chief of the Editorial

Board. Upon his death he was succeeded by Dr. William J. Mayo. For many years Dr. Mayo was in active charge of the Editorial Department. To his vision and judgment has been due the discriminating character of the editorials. Lately Dr. Donald C. Balfour has successfully assumed this responsibility.

Although the chronicling of scientific progress has been its primary objective, the journal has sought also to foster the cultural aspects of surgery and to honor those who established and perfected its principles. Under the supervision of Dr. Alfred Brown, "Old Masterpieces in Surgery" acquainted the oncoming generation with the history of surgery. Under the direction of Dr. William J. Mayo and Dr. Donald C. Balfour the department of "Master Surgeons" has recorded the achievements of the surgeons of the western hemisphere and aroused our pride in their accomplishments.

The journal is known and read wherever medicine is practiced. It has a large number of subscribers abroad and penetrates into little known centers in Asia, Africa, and the islands of the Pacific. The medical libraries of the world have its numbers filed as a permanent record of the advances in surgical knowledge during the fruitful period spanned by the publication of SURGERY, GYNECOLOGY AND OBSTETRICS.

It has been the desire of both Dr. and Mrs. Martin that after their deaths, the journal with its real estate should become by gift the property of the American College of Surgeons. Since the holdings of Dr. Martin at the time of his death in conjunction with the personal holdings of Mrs. Martin gave them complete ownership of the stock, their desire may be accomplished.

The regents of the College believe that this final service of Dr. and Mrs. Martin to the College and the profession may be made a worthy memorial to Dr. Martin's vision and ideals.

ALLEN B. KANAVEL.

FRANKLIN H. MARTIN AND THE AMERICAN COLLEGE OF SURGEONS

FRANKLIN MARTIN died Thursday, March 7, in Phoenix, Arizona, where up to a few days before his death he was occupied with the affairs of the American College of Surgeons, especially with preparations for the next Clinical Congress to be held in San Francisco in October.

We do well to pause to pay tribute to this great leader to whom more than to any other individual the American College of Surgeons is indebted for its foundation and its development during the twenty-two years of its existence. With courage, imagination, wisdom, unfailing loyalty to the best interests of the College and with rare executive ability, Dr. Franklin Martin as Director-General has guided its destinies.

It is fitting at this time to review briefly the history of the College—a history which will be his finest monument—for his name is indelibly inscribed upon each of its multifarious activities.

At the time the American College of Surgeons was founded, and earlier, many surgeons, particularly the members of the American Surgical Association had felt the necessity for an organization of the active surgeons which would standardize the practice of surgery. To this end at the Clinical Congress of Surgeons of North America held in New York in 1912, an organization committee was appointed. Thus the American College of Surgeons is the direct outgrowth of the Clinical Congress of Surgeons, a Congress which was originally fostered and promoted by Dr. Franklin H. Martin, who suggested the formation of this organization committee of the College.

Confronting this committee were two lines of procedure: (1) to follow the pattern of such an organization as the Royal College of Surgeons in England and admit members only on examination; or (2) to admit to Fellowship all the members of the sixteen principal societies representing surgery and its various specialties as a nucleus for the whole and then to add to that number all those surgeons in all parts of the United States and of Canada who by their training, experience, and achievements were surgeons in good standing.

The second of these plans was adopted by the organization committee and it at once became evident that unless the College were to grow very slowly it would be necessary to make a survey of the practicing surgeons throughout the country to determine who were qualified to become the first members of the College.

Such a survey was made, principally by Dr. Martin, who visited all the leading medical centers throughout the United States and Canada. As the result of this survey, four hundred and fifty prominent surgeons met in Washington on May 5, 1913, adopted the By-laws, rules, and regulations suggested by the organization committee and elected the first officers of the College, and the Board of Regents and Board of Governors. The original Board of Governors was comprised of the four hundred fifty founders of the College; but the By-laws provided for a permanent Board of one hundred fifty members, to be divided into three groups, fifty members to be elected by the Fellows at each annual meeting of the College. The government of the College was to be centralized in a Board of Regents whose decisions would be carried out by a Director-General. The recommendations for further Fellows of the College were to be made by credentials committees representing each state of the United States and each province of the Dominion of Canada. This plan was adopted at the organization meeting. Soon after the organization of the College it was decided that the College should endow itself by asking each Fellow to subscribe five hundred dollars, to be paid in a lump sum or by payments of not less than twenty-five dollars a year.

By this rapid start in membership and in financial support the College had a forceful momentum and was able at once to launch forth on its campaign to carry out its original purposes which are stated in the following summary of a clause of the Articles of Incorporation, published in each *Year Book:* "The object for which it is formed is to establish and maintain an association of surgeons, not for pecuniary profit, but for the benefit of humanity by advancing the science of surgery and the ethical and competent practice of its art."

It is my opinion that when the College was organized, there was no one man who could have equaled the performance of Franklin Martin as Director-General, for more than to any other one man, the credit belongs to him for the foundation of the College, its organization, for the rapid mobilization of surgeons to form its constantly increasing membership, the shaping of its policies, the solving of the many difficult executive problems which have repeatedly presented themselves. In addition to his many official trips throughout the United States and Canada, Dr. Martin has promoted the interests of the College in South America and in Australia by personal visits.

Throughout these twenty-two years, I have had the opportunity of observing the constructive genius of Franklin Martin at close range. I should say that except for his peculiar talents, the College would not be in its present position. Franklin Martin undertook every bit of the leadership that was delegated to him and often initiated new projects. The whole picture has constantly been one of rapid construction and of militant progress, as exemplified by the many activities of the College and by the work of its Department of Hospital Standardization; the Registry of Bone Sarcoma; the Committee on the Treatment of Malignant

Diseases; the Committee on Fractures; the Board on Industrial Medicine and Traumatic Surgery; the Department of Clinical Research; the organization of Cancer Clinics; the Regional Meetings; the annual Clinical Congress; the Library which is ready to serve each Fellow by literary research; and the Board on Medical Motion Picture Films.

In addition to a very high level of efficiency we have our property in Chicago which aggregates in value more than two million dollars—assets which include the endowment fund, land, buildings, and equipment.

The very characteristics which have made these achievements possible inevitably sometimes seem too aggressive; but in a formative period they are necessary even though they may arouse some antagonism. A lively imagination, courage, spirit of adventure, outstanding executive ability, great loyalty to a cause, can never be outweighed by lesser qualities. Such characteristics speak for themselves in outstanding achievements.

Dr. Martin not only was intensely loyal himself but was able to inspire loyalty in those associated with him as is demonstrated by the fact that the Editorial Board and the business personnel of SURGERY, GYNECOLOGY AND OBSTETRICS have remained practically unchanged since Dr. Martin founded this journal, with the exception of those called by death; and the outstanding characteristic of the working staff of the American College of Surgeons has been its long time loyalty to its Director-General. Those who knew Franklin Martin best, trusted him most.

All through his life, and despite every disguise, Franklin Martin has been shy, fearless, imaginative, idealistic, and a dreamer. Long will he be known among the great dreamers in medicine. He dreamed a dream, and the greatest surgical journal in the world was born; he dreamed again and the Clinical Congress of Surgeons of North America appeared; he dreamed yet again and the American College of Surgeons came into being.

GEORGE CRILE.

FRANKLIN H. MARTIN AND THE GREAT WAR

FRANKLIN H. MARTIN was, for half a century, one of America's distinguished surgeons; more than that, he demonstrated to an unusual degree the rare gifts of administrative genius and vision. It was logical, then, that when the clouds of war began to hover over our land, with characteristic foresight, as Secretary-General of the American College of Surgeons, he should tender the services of that organization to the Surgeon-General of the United States Army to aid in re-organizing and enlarging the Medical Reserve Corps. With the acceptance of this timely offer two thousand Fellows of the College, of military age, immediately enrolled in the Medical Reserve, and until the Armistice was signed this great organization of surgeons took the lead in every wartime medical program.

On August 29, 1916, through the enactment of the National Defense Act, President Wilson was authorized by Congress to appoint an Advisory Commission consisting of seven persons, especially qualified in their respective fields, to co-operate with the Council of National Defense in its task of mobilizing the resources of a nation dedicated to peace into a fighting force sufficiently powerful to stay a foe trained for centuries in the arts of war. Through the persistent endeavors of the Committee of American Physicians for Medical Preparedness, representing the American Medical Association, the Congress of American Physicians and Surgeons, the American College of Surgeons, the Clinical Congress of Surgeons of North America, and the American Surgical Association, totaling a membership of ninety thousand medical men, the President was convinced that the medical profession should be accorded a place on the Advisory Commission. The question then arose: who, among the members of the scientific medical profession, was qualified to fill this important and exacting post?

Franklin Martin was the unanimous choice of the committee and in October, 1916, he was notified of his appointment by President Wilson.

The Council of National Defense, consisting of six members of the Cabinet: Secretary of War, Newton D. Baker; Secretary of the Navy, Josephus Daniels; Secretary of the Interior, Franklin K. Lane; Secretary of Agriculture, David F. Houston; Secretary of Commerce, William C. Redfield; and Secretary of Labor, William B. Wilson held its first meeting in Washington on December 6, 1916, with the seven members of the Advisory Commission, Daniel Willard, president of the Baltimore and Ohio Railroad; Hollis Godfrey, president of Drexel Institute; Howard E. Coffin of Detroit; Bernard Baruch of New York; Julius Rosen-

wald of Chicago; Samuel Gompers, president of the American Federation of Labor; and Dr. Franklin H. Martin.

Commenting on the sentiments of the commissioners on this occasion, Dr. Martin said:

> We, as members of the Commission, had arrived in Washington with somewhat vague conceptions of our duties. We met the six members of the Council, who were equally vague as to our responsibilities, but we rapidly became acquainted. Each gave his idea of the requirements of the situation and of the immensity of the task that we must assume—and then we realized that our group, in joint action, would virtually become a War Cabinet, which should not only advise, but plan, execute, and direct the activities of our Government in its preparation for defense in the greatest war of history. No commissioners ever entered Washington at the call of a President with more humility and with less concrete knowledge of what they were expected to accomplish.

However, Franklin Martin shouldered the responsibilities of the post assigned him with characteristic eagerness, and immediately proceeded to formulate a program that would be operable when and if we were drawn into the world conflict. His department in the Council of National Defense was one of the first to establish a routine that functioned with clock-like precision until April 1, 1919. The outstanding men and women in medicine and the allied specialties in association with him gave the utmost of their talents and abilities to uphold the well-known devotion to service of the medical profession. Under his direction the medical resources of the nation were so adequately mobilized that a steady flow of men and materials perfectly synchronized as to supply and demand, was at all times available.

The General Medical Board of the Council of National Defense, of which Dr. Martin was chairman, included the leaders in medicine, dentistry, and nursing from civil life. This Board, with the Surgeons General of the Army, Navy and Public Health Service, and the chairman of the American Red Cross, guided the policies of the special committees composing the Medical Section of the Council of National Defense. These committees, more than a score in number, included the medical, dental, and nursing professions, and covered every angle of medical preparedness. In addition to the Washington organization, state and county committees co-operating with other defense agencies, extended their activities into every town and hamlet in the land.

As a result of this co-ordinated effort, the General Medical Board and its subsidiary committees, more than any other one agency, may be credited with the splendid showing of the medical profession in its voluntary enrollment for service. In November, of 1917, there were 440 medical officers in the Army and 329 in the Navy. At the armistice there were 30,591 medical officers in the Army, and 2,570 in the Navy. And in the Volunteer Medical Service Corps, an organization of physicians barred from active duty because of age, physical disability, or

home responsibilities, 72,219 enrolled. Likewise in the nursing and dental services, enrollments far exceeded the quota set for an army of five million men.

It is impossible in the limited space available here, to enumerate, be it ever so briefly, measures initiated by Dr. Martin, and the programs in which he participated during the war years. He worked assiduously to secure proper military rank for the doctors and nurses in the military services. He toiled unceasingly to supply the government with medical men and materials, to meet the demands of war. His own and foreign nations decorated him—and rightly so—for conspicuous service.

During the nearly two years of continuous association with Major-General William Crawford Gorgas, Surgeon-General of the United States Army, Dr. Martin came to know and admire this modest physician and ultimately a warm and enduring friendship developed between the two. After Gorgas' death in July, 1920, it was only natural that Dr. Martin should be one of a group who felt that this man was worthy of a memorial—not of stone or bronze—but a living vital organization which would embody the hope of freeing the world from disease—the object to which the life of Gorgas had been consecrated. As Surgeon-General of the Army, Gorgas instituted the policy of strict physical examination for all recruits with regular check-ups at intervals thereafter. This resulted in an army of physically fit men to meet the rigors of war and it is a well known fact that our soldiers maintained the highest health rate of all combatants. General Gorgas often said to Dr. Martin, "Wouldn't it be wonderful if some day a system of strict physical examination could be applied to our civil population?" The story of Gorgas' sanitary work in Havana and Panama is familiar not only to the scientific world but to the laity as well. So in planning the memorial to perpetuate his name, it was fitting that the two great works of Gorgas—personal health and sanitation—should be chosen as the basis on which the Gorgas Memorial should proceed. After the institution of an ambitious, nation-wide program in health education, it was characteristic of Dr. Martin's foresight that he should vision in the Gorgas Memorial Institute an opportunity for the creation of an international research center in which the countries of Latin America, where Gorgas had achieved his magnificent work in sanitation, should have a part. Out of this vision has grown the Gorgas Memorial Laboratory in Panama, already a recognized research center, underwritten by the United States Government, and destined to play an increasingly prominent part in the fight of science against disease.

Franklin Martin was above all a good soldier. He was brave; he was fearless; he would not recognize defeat, and once he had determined on a course, his generalship, untiring energy, and fighting force could be depended upon to see him through to a successful conclusion.

CARY T. GRAYSON.

FRANKLIN H. MARTIN—THE MAN

TO paint a word picture adequate of Franklin Martin, a man who as he matured became the greatest organizer which the past fifty years of medicine has produced, a man honored by his profession, decorated by his own and foreign governments, is indeed a difficult task. However, as it was my privilege for many years to enjoy his close friendship, to me the task becomes a labor of love.

There are many qualities common to those among men who leave their names conspicuously high on the rolls of achievement of the era in which they have lived. Oft included in these are the circumstance of lowly birth and early struggles against privations combined with integrity of mind, indomitable will, and other steadfast qualities. There are, however, deeper influences which must predetermine a man's ultimate worth, and nature having endowed Franklin Martin with the rugged qualities of strong character gave him also a fineness of sensibility which tempered his thoughts with keen discernment and his judgment with unfailing fairness and justice.

We see him in his youth with the spirit of D'Artagnan, recognizing no obstacles as insurmountable and ready for life's adventure with a happy heart. We see him passing through life retaining this happy heart and always sharing it with others. We see him at eventide with the spirit of youth and merriment still retained and then understand why it was he who wrote *The Joy of Living*, for life to him had been a joy. It was not alone the joy of achievement which for him produced satisfaction for no one labor was ever finished before his restless brain had conceived or he had had another thrust upon him. It was that joy which comes from doing for others which was the motivating force behind him. And the extent of his work for others is the true measure of Martin's greatness.

In each of the great activities in which he played such a major rôle it was his clear vision which insured their lasting success. Martin had the unusual combination of not alone being an organizer, but having the added quality of being able to make his organizations functionate peculiarly well after he had built them. As he was progressive in his thought, so were his organizations. Not only did they fulfill the objectives for which they were designed, but became active influences for advancement with changing progressive thought. This again was evidence of his farsighted vision.

The spirit of his soldier father, who sacrificed his life in public service during the Civil War, influenced Martin throughout his life. He was ever ready to aid

civic organizations, and took part in many projects to advance the welfare of Chicago. He was intensely loyal to his alma mater, Northwestern University, of which he was a trustee for a decade; and he was an active member of the various organizations which served its alumni. He gave to the U. S. Army Industrial College, the Fuel Conservation Institute, and the Board of Education of Chicago much time and thought. He was international in his outlook, and participated with enthusiasm in the deliberations of the Committee on Co-operation in Latin America, the Chicago Council on Foreign Relations, the American Academy of Political and Social Sciences, and the Italy-America Society.

Martin had an extraordinary ability to appreciate immediately which were the most important details of any problem and to recognize instantly the right thing to do under adverse conditions. There are many instances which I might relate which are indicative of this. One, however, stands out rather vividly in my memory. On a certain night shortly after the First Liberty Loan Drive had been started, he and I were at a Broadway theater. Between the acts a drive was staged and the men who were soliciting subscriptions were meeting with poor success. At that time Martin was interested in stimulating enlistments in the Medical Reserve Corps of the United States and the lukewarm reception which the audience were giving to the request for subscriptions disturbed him. He became restless and told me he must help the men on the stage stir up some enthusiasm for the Loan. He was dressed in his Colonel's uniform, and as he arose from his seat to offer his subscription, standing alone in that audience with his soldiery carriage, his chiselled face and flashing eye, he made an instantaneous effect on the audience, a sight never to be forgotten. He offered his subscription for the bonds and as the audience went wild with applause, not to be outdone by the soldier, they made the drive in that theater that night one long talked of. As he reseated himself beside me, he leaned over and whispered, "God knows how I'll ever pay for them," but he had saved the situation.

Martin will go down through the pages of American history as a courtly man, ever actuated by the strongest principles of high Americanism. He had the simple faith of his forefathers with a strong pioneer spirit. Whenever he fought for his beliefs, he was a foeman worthy of his adversary's steel. He was a rare combination of stern forcefulness and sympathetic kindliness, and I believe no more fitting description of Martin as a man could be made than by a summing up in two words: Franklin H. Martin—Surgeon and Gentleman.

"Sunshine was he in the winter day,
And in the midsummer, coolness and shade."

J. BENTLEY SQUIER.

BIOGRAPHICAL OUTLINE

Born in Ixonia, Wisconsin, July 13, 1857; son of Edmond and Josephine (Carlin) Martin
Died in Phoenix, Arizona, March 7, 1935
Educated in public schools and academies of Wisconsin
Northwestern University Medical School, M.D., 1880
Interne, Mercy Hospital, Chicago, 1880–1881
Member of Staff, South Side Dispensary, 1883–1888
Married Isabelle Hollister (daughter of John Hollister, a founder of Northwestern University Medical School) May 27, 1886
Professor of Gynecology, Policlinic of Chicago, 1886–1888
Organized, with Dr. W. F. Coleman, the Post-Graduate Medical School and Hospital of Chicago, 1888
Gynecologist, Woman's Hospital, Chicago, for many years, from 1887
Organized the Charity Hospital, Chicago, 1889
Organized SURGERY, GYNECOLOGY AND OBSTETRICS, 1905; the INTERNATIONAL ABSTRACT OF SURGERY, 1913; Editor-in-Chief, 1905–1935
Organized the Clinical Congress of Surgeons of North America (now the Clinical Congress of the American College of Surgeons), 1910
An organizer of the American College of Surgeons, 1913; Fellow, Regent, and Director-General, 1913–1935; President, 1929
Trustee, Northwestern University, Chicago, 1921–1931; Medical Counsellor, 1929–1935
Member, Advisory Commission of the Council of National Defense, 1916–1921
Chairman, General Medical Board, Council of National Defense, 1917–1918
Colonel, Medical Corps, U. S. Army, during period of Great War, 1917–1919; with A.E.F. for three months
Founder, and Chairman, Board of Directors, Gorgas Memorial Institute of Tropical and Preventive Medicine, 1921–1935
Honorary Adviser, United States Army Industrial College, 1925–1935

MEMBER OF MEDICAL AND OTHER ORGANIZATIONS

American Medical Association; Chairman, Section of Gynecology and Abdominal Surgery, 1895
American Gynecological Society; President, 1919; Member of Council, 1919–1922
Southern Surgical Association
Congress of American Physicians and Surgeons
Chicago Gynecological Society; President, 1894
Association of Military Surgeons
American Medical Editors' and Authors' Association; Member, Board of Governors
American Society for the Control of Cancer
American Hospital Association
Northwestern University Alumni Association; President, 1931; Director, Division on Medicine, 1921–1923
Northwestern University Associates
American Legion, Hyde Park Post
Chicago Literary Club
American Association for the Advancement of Science

DECORATIONS, HONORARY DEGREES, AND MEMBERSHIPS

LL.D., Queens University, Belfast, 1925; University of Wales, Cardiff, 1928; University of Pittsburgh, 1933
D.P.H., Detroit College of Medicine and Surgery, 1926

D.Sc., Northwestern University, Evanston, 1927
Companion of the Order of St. Michael and St. George (C.M.G.), 1919, in recognition of services rendered to the British Empire during the Great War
Distinguished Service Medal, U.S., 1926, in recognition of services rendered during the Great War
Commander of the Order of the Crown of Italy, 1931; in recognition of aid in founding Loyola Base Hospital, which served the fourth and sixth armies on the Italian front
Honorary Fellow, Sociedad Peruano de Cirugia
Honorary Fellow, Academia Nacional de Medicina de Rio de Janeiro, Brazil
Corresponding Member, Sociedade de Medicina e Cirurgia de Sao Paulo, Brazil
Corresponding Member, Sociedad de Cirugia, Buenos Aires, Argentina
Corresponding Member, Academia Nacional de Medicina, Buenos Aires, Argentina
Honorary Member, Sociedad de Obstetricia y Gynecologia, Buenos Aires, Argentina
Honorary Member, Eta Chapter, Alpha Kappa Kappa Fraternity

BOOKS AND MONOGRAPHS

Electricity in Gynecology, 1890
Treatment of Fibroid Tumors of the Uterus, 1897
Treatise on Gynecology, 1903
South America, from a Surgeon's Point of View (First Edition, 1922; Second Edition, 1927)
Gorgas, 1924
Australia and New Zealand, 1924
The Joy of Living, an Autobiography, 1933
Fifty Years of Medicine and Surgery, 1934
Digest of the Proceedings of the Council of National Defense and the Advisory Commission during the World War (Published by the U. S. Government as an official document), 1934